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AUTHOR	Young, Francis A., Ed.; Lindsley, Donald B., Ed.
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ABSTRACT

This book brings together papers presented at a conference on early experience and visual information processing in perceptual and reading disorders sponsored by the National Academy of Sciences. The goal of the conference was to integrate basic knowledge of structure and mechanisms of eye and brain with their function and their behavioral roles in perception, with the focus on underlying factors that may contribute to reading disorders. The first section discusses topics related to the role of the visual system: optical and oculomotor, retinal, and central neural factors. The second section discusses attentional and perceptual mechanisms. The third section discusses early experience and learning in visual information processing. The fourth section discusses the role of information processing in perceptual and reading disabilities. The fifth and final section discusses management of children with perceptual and reading disabilities. The introduction is written by Donald Lindsley and Francis Young. Individual papers are written by such authors as Jeanne Chall, Robert Boynton, Robert Doty, Roger Sperry, Richard Jung, George Sperling, Ira Hirsh, Merton Flom, William Mason, Robert Fantz, and H. Burtt Richardson, Jr. (TS)

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EDITED BY FRANCIS A. YOUNG / DONALD B. LINDSLEY

Early Experience and Visual Information Processing in Perceptual and Reading Disorders

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Proceedings of a Conference held
October 27–30, 1968, at Lake Mohonk, New York,
in association with the
Committee on Brain Sciences,
Division of Medical Sciences,
National Research Council

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Foreword

Lake Mohonk Mountain House has a history of hosting altruistic meetings long before they became so popular. In 1883, the first conference of Friends of the Indians was held there, a conference that later was broadened to include a more general subject: "Lake Mohonk Conference of Friends of the Indian and Other Dependent Peoples." In 1895, a conference on international arbitration met there. The dismal history of these subjects since then testifies that more than motivation and intelligence are needed to transform successful conferences into action.

More than twenty years ago, it was obvious that, given sufficient funds, scientists and engineers could put courageous men on the moon because these efforts would not be hampered by superstition. Problems of brain and behavior are another matter and in the opinion of many people constitute a most important frontier. Although there have been no comparable dramatic and highly visible breakthroughs in the knowledge of the mechanisms of childhood development, there has been a quiet revolution in the knowledge of how the child develops. It is now recognized that from birth to three years is an age of sinister importance. The hope is that similarly objective studies of how development proceeds will be extended (and supplement existing observations of the period) to seven years of age, when the child enters public life in the school situation.

It has been apparent since 1960 that cognitive development is not dependent on motor accomplishments. It might seem to be only a small step from observation of overt actions in infants three to six months old to careful observations of visual attention to various objects at a few days of age, but the results have been revolutionary in directing attention to the schemata of cognitive development at the earliest age. Instinctual behavior in human infants may be considered minimal: the sucking reflex, the crying signal, and perhaps imitative reactions, early body playing, and infantile sexuality belong in this category. The quiet revolution of the last decade has taken infancy out of the realm of concern about primary necessities of vegetation and into the sphere of the fully living human being and has shown that cognitive learning and socialization can begin at a very early age. Parents who have the interest and ability to understand its importance have accomplished this transition in child-rearing fairly well on a naturalistic, almost subconscious, level. Those children whose parents are too harried by economic or other problems are not so fortunate.

The conference speakers put the problem in bold relief. Although the research studies described emphasize the importance of exteroceptive stimulation of the infant, they by no means derogate the importance of somatosensory and motor functions and loving maternal and paternal care. The capacity of the very young infant to find intellectual pleasure in manipulating a mobile, for example, was documented, but the infant can and often does receive similar "education" from an old tin can if there is the emotional security of parental care and affection.

The conference was notable for its scientific objectivity. The goal was to bring people of diverse disciplines together to communicate on the role of early experience in visual information processing. Discussions were confined to the subject with no "practical" implications for belief-system conditioning techniques. The general tone was free of acrimonious arguments, as if each participant knew that the issues were too important to permit personal polemics. Even during the extracurricular hours, when individual differences of opinion were more openly expressed, the arguments, although pointed, were urbane and friendly. There was a remarkable demonstration of the free-inquiry approach to complex problems. The data were the result of hardheaded and disciplined naturalistic observations gently and skillfully guided by instrumented measurements and manipulation of the environment. Freud, Watson, Gesell, Piaget, and many other pioneers were present in the background, obviously inspiring or provoking the investigators, but never dominating the scene.

I was impressed by the emphasis on the necessity of integration of two or more sensorimotor facilities. One wonders about the phenomenon of Helen Keller and is led to the speculation that a facility not properly used may have a negative effect on other facilities. This suggestion may answer some of the puzzles presented.

Appropriate attention was paid to the handicapped child, but what stood out was the need for greater understanding of "normal" or near-normal developmental problems; studies of "normals" give great insight into the problems of the marginal child. This volume of proceedings is a valuable supplement to the three-year study of the Joint Commission on Child Mental Health, which represents only one of many large-scale attacks on the problems of child development now receiving wide public attention, and it lends perspective to the work of the Interdisciplinary Committee on Reading Problems and the National Advisory Committee on Dyslexia and Related Reading Disabilities.

More was brought out at the conference than is immediately apparent. If the child from birth to three years of age can gain such emotional and intellectual satisfaction from simple stimulation and coordination of visual, auditory, and somesthetic senses, do we not have a possible means for developing an adult with more adequate coordination of the phylogenetically older and newer parts of the brain? A by-product of the information gained from study of infancy and childhood may be the detection of autistic children at an age when remedial measures may be effective. Likewise, specific learning disabilities may be detected in time to institute effective measures.

WADE H. MARSHALL

Preface

The initial planning for this conference was done by the Committee on Brain Sciences* of the National Research Council. The Committee's mission has been to encourage a truly holistic approach to the study of brain and behavior: to communicate not only across disciplinary borders but also between the different levels—theoretical, basic, and applied—of research endeavor. In spite of difficulties inherent in successfully implementing this concept, the proposed conference received enthusiastic sponsorship by the Committee, and a planning group was appointed, consisting of Donald B. Lindsley, David Bodian, Eugene Roberts, and Francis A. Young.

The participants were drawn from three groups: experimentalists doing basic research in vision, audition, perception, and other cognitive functions; practitioners examining, diagnosing, and treating children with reading and perceptual disabilities; and educators concerned with the best way to teach children to read. The experimentalists were trained primarily as psychologists, neurophysiologists, and neuroanatomists.

*Members of the Committee at that time were Carl Pfaffmann (Chairman), David Bodian, Victor Denenberg, Edward Evarts, Ralph Gerard, Seymour Kety, Donald Lindsley, Neal Miller, Frank Morrell, Wilfrid Rall (from June 1968), Eugene Roberts, Walter Rosenblith (to June 1968), Francis Schmitt, and Klaus Unna.

The practitioners were ophthalmologists, neurologists, optometrists, and pediatricians. The educators were working in graduate schools of education and were training future teachers, in addition to carrying out research.

The goal of the conference was to integrate basic knowledge of structure and mechanisms of eye and brain with their function and their behavioral roles in perception, with the focus on underlying factors that may contribute to reading disorders. The speakers were urged not to talk for their colleagues in their own fields, but to emphasize points about which workers in other disciplines should be aware. This volume contains both the prepared manuscripts and the discussions that took place at the conference. Because it was often impossible to avoid the use of specialized terms, a glossary is included. The introduction includes a description of the visual process and other information thought to be useful to readers trained in disciplines peripheral to those of the speakers. This volume is intended for an interdisciplinary, scientific readership of wide range. It is hoped that readers, like the conference participants, may find new concepts and stimulation from this attempt to open avenues of approach to several important problems of childhood.

In an effort to reach the widest possible readership, publication of a consecutive account of the conference is being prepared by one of the editors in nontechnical terms with germane background material.*

Many factors contributed to the conference and to this publication. Crucial were the financial support of the conference by the National Institute of Neurological Diseases and Stroke and the subsidy from the Blaauw Fund of the National Academy of Sciences for publication of the proceedings. We thank the many persons who helped with the conference and the publication.

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*_Seeing, Perception, and Reading_, by Francis A. Young (to be published).

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DONALD B. LINDSLEY / FRANCIS A. YOUNG

Introduction

The principal means of acquiring and accumulating information is through the printed word or symbol. To optimize the use of printed information through a progressive learning process, it is necessary for a child not only to learn to read, but to gradually develop a high degree of skill, speed, and efficiency in reading.

Some children, known as nonreaders, never learn to read. Others learn to read, but only very slowly, with great difficulty, and inefficiently, thus impairing their acquisition of knowledge during the school years and throughout life. Still others learn to read on schedule, but with varying degrees of skill and efficiency, as measured in terms of speed of reading, comprehension, ability to select information and process it into memory storage, and ability to retrieve information from short- or long-term storage and use it for concept formation, thinking, and problem-solving. Of great importance, also, even in a child who has learned to read, are the ease of reading and the pleasure afforded by the reading process when correctly and efficiently learned and the rewards that it brings by way of achievement. Motivation to read and extend one's knowledge is crucial in the acquisition of education, culture, and general enlightenment.

It should be evident that reading, which, once learned, seems like such a simple process, is indeed a complicated aspect of a broader information-processing function, including perception and higher cognitive processes. The ability to see and read printed words depends on the qualities of the physical stimulus (e.g., illumination, visibility, contrast), the form and pattern of the printed elements (letters, words, sequences of words, and so on), and, of course, knowledge or understanding of the language of which the words are symbols. In addition, the eyes must be trained to scan the stationary stimulus patterns provided by the printed text in a series of stop-and-go movements, usually two or three fixations (saccadic eye movements) per line, affording tachistoscopic exposures during which the span of apprehension permits intake and processing of information, provided that attention is maintained. At some level of the visual system, either in the retina or farther along in the visual pathways of the brain, suppression or inhibition of input apparently occurs during the intervening eye movements, inasmuch as no smearing or blurring of the type is apparent during the course of scansion of each line. Likewise, the adjacent portions of other lines, although within the visual field of each fixation, are somehow suppressed, possibly by selective attention to the line being read.

These are just a few of the aspects of the process of reading with which the conference was concerned. In planning the conference, it was recognized that there is a great deal of basic information about vision and visual perception and that in recent years much new information has been acquired about the mechanisms of the eye and the brain that has not been brought to the attention of those concerned with applied problems in which these mechanisms are involved. Furthermore, considerable effort has been expended in a search for the onset and development of various psychologic and behavioral functions, as well as their anatomic and physiologic precursors. In some instances, deliberate interference with or blocking of development of functions has been attempted in young animals. In others, manipulation of the environment, either in the form of sensory deprivation or restriction or in the form of enrichment and enhancement of stimulation, has been studied, with the aim of determining how these changes affect the developing and maturing organism.

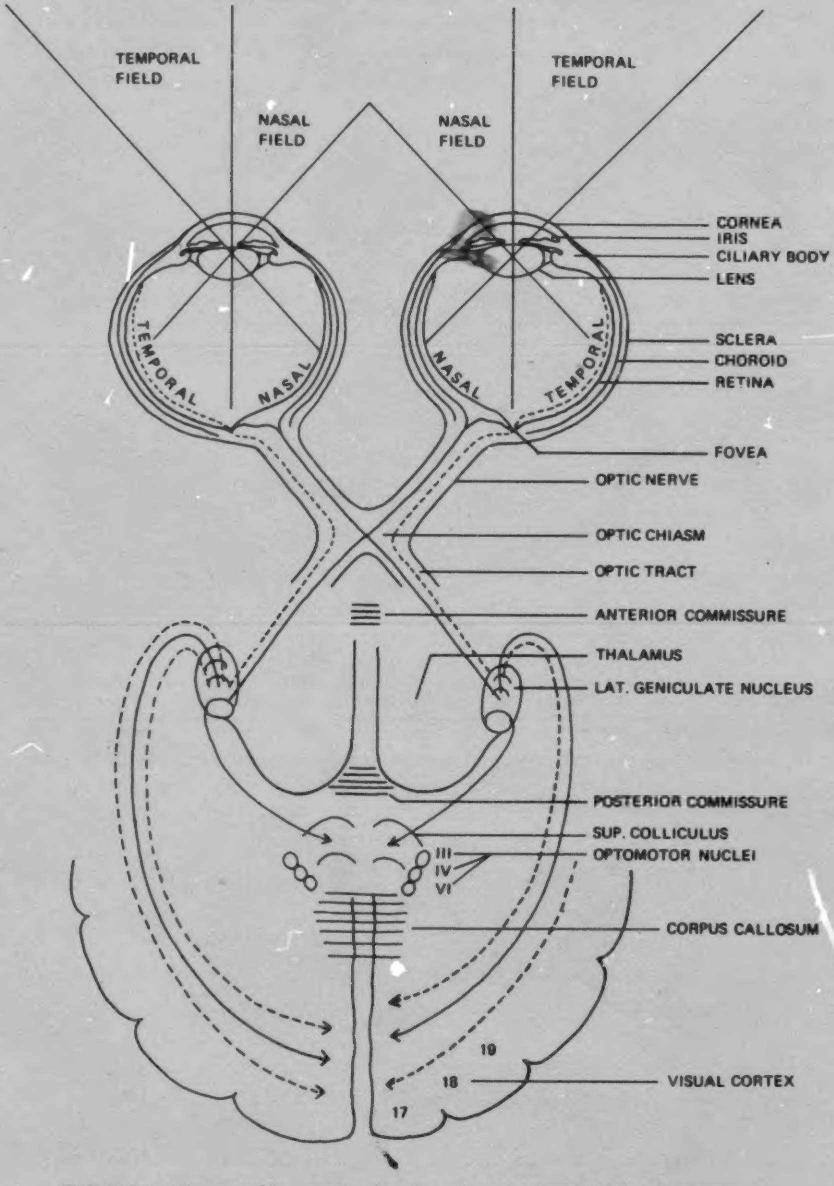
To provide an orientation toward reading behavior, Jeanne Chall reviews the philosophies and methods that have been used during the last 40 years in the teaching and assessment of reading skills. Trends in the teaching of reading have gone full circle from a "decoding emphasis"—

first learning the alphabet and the composition of words–to "meaning emphasis"–largely ignoring the alphabet and syllable approach and concentrating on the meanings of words and phrase patterns as wholes. During the last decade, the more innovative approaches have shown a swing back to decoding emphasis, to be followed when appropriate by meaning emphasis. Although the results of her study seemed to favor this approach, she cautiously states that the evidence is by no means clear-cut.

Dr. Chall's presentation emphasizes the importance of periodic conferences of this kind in extending the knowledge of reading and reading disorders and providing bases for decisions. It also highlights the too common failure of basic scientists to realize the extent to which their own somewhat specialized research may have important implications for educators and practitioners.

The initial papers are concerned with the role of the visual system in information processing, starting with the more peripheral aspects–those involving the eye–and moving along the visual pathways to the visual cortex. Because the nonspecialist may not be familiar with the visual process, a description of how the image is formed and what happens to the nerve impulses generated is presented here.

Figure 1 is a schematic illustration of the general features of the visual system. The eye is essentially a spherical body, except for the slight bulge at its anterior surface provided by the cornea, the exposed part of the eye. The lateral and posterior walls of the eyeball consist of the sclera, a tough outer membrane that serves as a protective and restraining covering for the contents of the eye, which are under pressure greater than that of the atmosphere. Inside the sclera is the deeply pigmented choroid, which contains the principal blood supply for the eye and prevents light from entering the eye through the sclera, thus preventing light scattering within the eye. Inside the choroid is the retina, containing the receptor elements (rods and cones); bipolar cells, which form synapses with cones and multisynaptic contacts with rods; and ganglion cells, which form synaptic contacts with bipolar cells. The axons of ganglion cells comprise the half-million fibers of the optic nerve in man. Also within the retina are important multiconnector cells (horizontal and amacrine), some cells thought to serve integrative and summative functions, and others thought to serve the role of lateral inhibition and excitation, which are important to image contrast in the retina and to on- and off-center receptive fields. The rods and cones are next to the choroid.


TEMPORAL FIELD
NASAL FIELD
NASAL FIELD
TEMPORAL FIELD
TEMPORAL
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NASAL
TEMPORAL
CORNEA
IRIS
CILIARY BODY
LENS
SCLERA
CHOROID
RETINA
FOVEA
OPTIC NERVE
OPTIC CHIASM
OPTIC TRACT
ANTERIOR COMMISSURE
THALAMUS
LAT. GENICULATE NUCLEUS
POSTERIOR COMMISSURE
SUP. COLLICULUS
III
IV
VI
OPTOMOTOR NUCLEI
CORPUS CALLOSUM
19
18
17
VISUAL CORTEX

FIGURE 1 Diagram of human visual system, showing visual fields and pathways.

Light penetrates the cornea and passes through the pupil, which is formed by the iris. The iris, through the action of its circular and radial muscular coats, regulates the size of the pupil and the amount of light entering the eye. The entering light is refracted or bent primarily at the air–cornea interface and slightly at the interface with the aqueous humor of the anterior chamber immediately behind the cornea. Following the major refractive effect of the cornea, the lens, whose curvature increases when the suspensory ligaments that encompass it are relaxed by action of the muscles of the ciliary body, provides the second most important refractive effect on the light rays. Light continues through the gel-like vitreous humor filling the posterior two thirds of the eyeball and then passes through the layer of ganglion cells and their axons, which leave the eye through the optic disk (blind spot) to form the optic nerve. The light must also penetrate the layer of bipolar cells before it reaches the absorbing substances contained within the rods and cones, where photochemical processes occur. These reactions to light cause generator potentials to be built up in the receptor cells, thus initiating nerve impulses that are transmitted across the synapses between receptor cells and bipolar cells and then across the synapses between bipolar and ganglion cells. Horizontal and amacrine cells play a role in regulating the flow of impulses through these retinal paths to the optic nerve. For the most part, light that escapes the retinal receptors is absorbed by the heavily pigmented choroid. A schematic illustration of neuroretinal components is provided in the paper by Boynton (Figure 8, p. 107); histologic sections of the retina are reproduced in Glickstein's paper.

The optical apparatus of the eye needs physical and dynamically functional characteristics for bringing patterned rays of light into proper focus on the sensitive and labile photographic screen known as the retina. Typically, if a point source is to be seen clearly at reading distance, accommodation must occur and the eyes must converge (as described below) in order to bring the image of the point onto the fovea (the portion of the retina at the back of the eyeball where only cones are found). At the same time, pupillary constriction limits the exposed lens to its region of greatest curvature and least aberration. The high density of cone receptors in the fovea and their essentially one-to-one relationship to bipolar and ganglion cells affords a high resolution of the image projected on the foveal retina. If light were focused parafoveally or on the peripheral retina, fine discrimination and high-acuity resolution would not be possible. Such a situation may arise in the case of strabismus or "squint," described in Alpern's paper, if the muscles of the eyes are inadequate to maintain proper convergence alignment, so that the images in the two eyes are not formed on corresponding retinal points. In normal eyes, the two images are formed on corresponding retinal points, and the slightly different views of an object give rise to the stereoscopic effect of depth. However, with respect to most plane surfaces, the image formed on the retina of the nondominant eye is partially (sometimes wholly) suppressed in favor of the dominant eye. This suppression may be demonstrated by holding a finger at arm's length and looking over its tip binocularly at the wall beyond; when the right eye (if dominant) is closed, the view with the left eye shifts markedly to the right, whereas, if the left eye is closed, the view remains the same as when seen binocularly.

Visual accommodation is the process by which the lens of the eye adjusts to focus light from a near object on the retina for near-point viewing. As the ciliary muscles attached to the ciliary body contract and reduce tension on the suspensory ligaments, the elastic properties of the lens allow it to bulge anteriorly; it thus has greater curvature and refracting power to bend light rays to a focus on the retina for a near-point fixation. When the eyes converge on a more distant point, the ciliary muscles relax, tension of the suspensory ligament is restored, and the lens tends to be flattened out, with less curvature and refracting power. When either (or both) of these actions is no longer possible or when other characteristics of the eye change, making it impossible to bring light to a focus on the retina, corrective lenses are necessary. If light comes to a focus behind the retina, rather than on it, owing to lack of refractive power of the eye, a condition of hyperopia, or farsightedness, exists. In infancy, although the eye itself is small, the lens is nearly of adult size and thus contributes a greater portion of the total refraction of the eye than in the adult. In old age, the total refracting power of the lens is reduced by decreased elasticity, producing hyperopia, or farsightedness (presbyopia). Light focuses in front of the retina in nearsighted, or myopic, persons, who have no difficulty reading at near distances that would be difficult for a normal (emmetropic) eye. The power of a lens necessary to correct for improper focus on the retina is expressed in diopters, or the reciprocal of the focal length (given in meters). Thus, a concave (minus) lens with a focal length of –0.25 m would be 1/–0.25, or –4 diopters.

Other aberrant properties of the optics of the eye that may lead to difficulty in reading come under the heading of astigmatism. The curvature of the cornea or lens may be irregular in one or more dimensions, so that the image on the retina is clear in one place and fuzzy in another.

During accommodation and convergence, the pupil constricts. All three of these actions are mediated by the third cranial nerve (oculomotor); they occur in focusing on a line of type in reading and create a sharp image on corresponding points of the two retinas. In reading, the eye moves from left to right in quick saccadic jerks produced by the external rectus muscle of the right eye and the internal rectus of the left eye, while the opposed recti of both eyes retain sufficient tension to cause proper convergence. A pair of muscles is also attached above and below the eyeball. These four symmetrically placed muscles control up and down and right and left movements of each eye. In addition, a pair of obliquely placed muscles rotates and tilts the eye downward or upward. Innervation of the external eye muscles is via the third, fourth, or sixth cranial nerve. It is believed that in voluntary and perhaps also involuntary eye movements motor discharges from the motor eye fields of the cortex (Brodmann's area 8) convey impulses to the region of the midbrain and pons. Thus, cortical control of eye movements is possible, but it is uncertain how much of a role the higher centers in the motor cortex play in the usual tracking movements of the eyes during reading, with frequent stop and go interactions. It seems more probable that tracking movements are mainly of reflex nature and are a function of retinal excitation and neural discharges feeding back to optomotor and pretectal nuclei.

When light activates the retina and generates impulses in the optic nerve and optic tract, most of these neural messages pass to the lateral geniculate nucleus in the thalamus and thence via the optic radiations to the primary visual cortex (area 17) along the striate area of the medial surface and the tip of the occipital lobe. A much smaller fraction of the impulses pass by way of the superior colliculus to the tectal region of the midbrain. In Figure 1, pathways along the superior colliculus are shown schematically as curved lines emanating from below the lateral geniculate nucleus and passing to the superior colliculus or pretectal nuclei, whence it is a short path to the optomotor nuclei and the reticular formation of the lower brain stem. The reticular formation plays a role in arousal, activation, and attention; some investigators think that it also plays a role in learning and memory processing. Thus, in addition to the specific visual pathways via the lateral geniculate nucleus of the thalamus and optic radiations to the visual cortex, there are indirect or nonspecific pathways from the optic tract to the superior colliculus and pretectal nuclei of the midbrain. The latter undoubtedly constitute important feedback pathways for the regulation of the optics of the eye and eye-movement control, and provide possible connections with the reticular formation of the lower brain stem, and possibly also with the pulvinar. The excitability of the cortex may be influenced through these nonspecific arousal and activation mechanisms. Thus, such indirect control of cortical activities generally, and perhaps visual cortical activity specifically, may play a role in the degree to which attention is maintained in reading and similar visual tasks. The nonspecific visual pathways are described in detail in the paper by Buser.

The lateral geniculate nucleus is organized as a six-layered structure, and a pattern of input from the two eyes comes to the lateral geniculate laminae in a partially alternating manner. The organizational relationship to function in the retina and geniculate is demonstrated in Glickstein's paper. The receptive-field concept, described in detail in the paper by Jung and Spillmann and first demonstrated in the retina, includes the idea that the field representation is partially retained and in some respects elaborated at the level of the lateral geniculate nucleus and the visual cortex. In the visual cortex, organizations of neuron units seem to represent simple, complex, and hypercomplex patterns of activation and functioning with respect to information presented to the retina. Some units respond to angular and linear stimulus arrays, others seem to respond only to movement, and so forth. Marg reports on some of these characteristics for single units of the human visual cortex.

Finally, because it has particular relevance to Sperry's paper, attention is drawn to how the nasal and temporal halves of the retina receive stimuli from the temporal and nasal halves, respectively, of the visual environmental fields. The optic nerve fibers from the nasal halves of each retina cross to the opposite sides in the optic chiasm and in the optic tract join the fibers coming from the temporal half of each retina. Thus, the temporal half of the left retina and the nasal half of the right retina, each viewing part of the right-half nasal field for left eye and temporal field for the right eye of the field of view when the eyes are converged to a fixation point, send their messages via pathways to the left hemisphere of the brain (left visual cortex of the occipital lobe). Similarly, the nasal half of the retina in the left eye and the temporal half in the right eye send their messages via pathways to the right hemisphere of the brain. A longitudinal (midsaggital) cut through the optic chiasm would eliminate the temporal visual fields for both eyes by inactivating the pathways leading to the visual cortex from the nasal half of each retina. Argument still prevails as to whether and how the central area of the fovea divides its input to the two halves of the brain, or whether the fovea centralis is bilaterally represented in the visual cortex of both hemispheres.

It is generally assumed that the left hemisphere of the brain in right-handed (and perhaps right-eyed) persons is the dominant or major hemisphere, whereas the right hemisphere is the nondominant or minor hemisphere. Objections to this assumption usually revolve around the determination of laterality or sidedness and whether hand or eye use is an adequate index of "native" or hereditary tendencies to unilateral hemisphere control or whether eye or hand use merely reflects the fact that we live in a world better adapted to the right-handed and right-eyed majority of people. For example, a typical school chair with a writing arm on the right is awkward for a left-handed person. There are many other examples of both unimanual objects and uniocular instruments built for the predominantly right-handed population. It is argued that such a dextrally organized world tends to prevent a hereditarily determined preference for sinistrality from becoming a reality. It has frequently been observed that so-called right-handed children may have a native preference for the left that manifests itself on some unimanual or uniocular tests that are not biased by learning in favor of the right. Similarly, some left-handed children who were compelled to write with their right hands may have developed stuttering speech, illegible writing, or other aberrations. The tendency to read or write letters and words in reversed or mirror fashion appears in some children with reading disabilities.

And there is much evidence that the controlling motor speech center (Broca's area) is situated in the dominant hemisphere, namely, in the left hemisphere of right-handed subjects. Damage to this area usually induces a motor aphasia, whereas damage to the homologous area of the nondominant hemisphere does not have this effect. Thus, for speech, there seems to be good evidence of unilaterality of control. Sperry's results in contrasting the perceptual capabilities of patients with partially "split" brains, through section of the corpus callosum, after a lifetime of usage and orientation in which one hemisphere presumably dominated showed greater differential hemispheric effects and more profound deficits than in patients with agenesis of the corpus callosum. The latter patients used the two hemispheres equally, often apparently without bias due to hereditarily or environmentally determined dominant hemispheric tendency; the hemispheres tended to have equivalent perceptual response patterns when tested separately, that is, by limiting input to one side or the other. By contrast, the group with section of the corpus callosum showed more complete visual perceptual performance for the dominant hemisphere than was possible with the nondominant hemisphere, but the minor hemisphere seemed to handle some nonvisual performances that the major hemisphere did not. These results will, of course, have to be confirmed and extended, but they are important to concepts about the conflicting tendencies, with respect to both sensory input and motor outflow, that interfere with such processes as reading, writing, speaking, and, probably, general motor coordination in children or adults.

There may also be important implications in this work for the establishment early in life of sure laterality and hemispheric dominance. Once it is determined that a child is right- or left-handed and has a definitely dominant hemisphere for sensory input and motor outflow, there may be stimulus patterns or situations that should be reinforced if the dominance bias is to be retained and strengthened. There may even be characteristics of printed or written words that are not appropriate for persons with sinistral organization and dominance of the brain. This would seem to be true: the favored extensor usage of the arm in writing tends to favor the forward-moving (to the right) tendency of writing letters and words. That is to say, some letters—such as "d" and "q"—perhaps require that the forward abduction (extensor) movement to the right be stopped in order to backtrack with an adductor (flexor) movement of the arm in making the bulge of the letter, whereas the writing of "p" or "b" seems mainly consistent with this direction of movement of the arm. More of our practiced movements with the arms, both reflex and voluntary, seem to involve extensor and abductor (push-away) movements than flexor and adductor movements. There is room for much more research in this field, and it seems apparent that such research will have relevance for speaking, reading, and writing, not to mention other functions that depend heavily on these basic modes of communicating and processing information.

It is apparent that reading involves physical environmental stimuli whose potential informational value can be realized only if the visual system is capable of carrying the load that it is presumed to carry. Its optical and oculomotor properties and characteristics must be properly functioning and understood to achieve optimal conditions for reading.

Francis Young has studied several visual measuring methods in populations living and working under environmental conditions that have influenced the manner in which their eyes handle refractive problems. His data on nonhuman primates, Eskimos, and other people (both children and adults) appear to him quite compelling in showing that environment and usage of the eyes have strong influences on their optical characteristics. He reports that there are studies that favor hereditary determination of optical characteristics of the eye and others that favor environmental influences, but that the evaluation methods are not always appropriate and the data in many cases have been obtained under an existing situation, rather than experimentally. Young proposes several refinements in the measurement of visual acuity and related optical characteristics of the eye. He believes that experimental studies in animals and man can be considered adequate for making evaluations only when several of these measures have been carefully applied and that the likelihood that children will experience visual difficulty due to optical causes in connection with reading can be appraised only when adequate assessment of schoolchildren is made by some of these methods.

Schubert, whose contribution is closely related to that of Young, attempted to create myopia and astigmatism by the use of lenses and to study the effect on far-point perception when the stimuli were presented tachistoscopically. He concludes that this experimental approach demonstrates that students with relatively mild degrees of myopia or astigmatism are likely to be handicapped in the school situation, where rapid presentations of material at varied distances are involved. It would seem that further experimental studies, closely related to classroom conditions, would add valuable knowledge about visual requirements for students in the school situation, especially in relation to reading.

Man's two eyes tend to work as a single unit because of their precisely coordinated movements. Cogan and Wurster discuss the development of the ocular motor control of the extraocular muscles that move the eyes. Assuming that a person has developed normal eye-movement patterns, how are these eye movements related to what is received in visual perception? This subject is discussed by Gaarder.

With normal functioning of the individual eyes and coordinated functioning of the two eyes as a unit, the organism is ready to receive visual stimuli and to bring them to a focus on the retina itself. On the retina, however, there must be sufficient variation in brightness in the stimulus pattern to permit the observer to discriminate the pattern. Without different levels of retinal contrast, all visual input would be uniform and consist mainly of evenly illuminated fields. The mechanisms underlying retinal contrast are discussed by Boynton. If the stimulus patterns overlap, binocular interaction may momentarily suppress retinal sensitivity. Alpern relates this situation to the pupillary reflex to light in strabismus.

Visual information reaching the brain is further processed by attentional and perceptual mechanisms in visual information processing. Doty discusses the various parts of the visual cortex and input areas of the nervous system that control input channeling and determine the characteristics of visual input, as far as the brain level is concerned. Sperry discusses cerebral dominance and its major role in maturation of the individual's perceptual behavior.

The papers mentioned previously deal with the characteristics of the visual input and the role of the nervous system in receiving and channeling the input. Turning now to attentional and perceptual mechanisms, the size of the visual receptive fields and the integration of perceptual phenomena to form a meaningful unit are discussed by Jung and Spillmann. Marg's findings from single-unit recording in man are relevant here. The retention and storage of visual perceptual input and its modification are the subjects of Sperling's paper, and he emphasizes the importance of scanning and rehearsal. Hochberg proposes that attention is an integral part of the reading process, and he explores the way in which the reader's knowledge affects paying attention. The importance of the relationship of language comprehension and use to reading ability is stressed by Hirsh.

Several papers consider the effects of early experience and learning on visual information processing, conditions that tend to vary from individual to individual, in contrast with the visual mechanisms related to receiving, channeling, organizing, and storing, which are relatively constant for all individuals. Riesen discusses the effects of various types of visual environments on the functioning of the retina itself, and Flom discusses ways in which experience influences the visual behavior of the developing infant and also considers the role of early environment on the development of anomalous visual behavior. Fantz and Lipsitt discuss the development of pattern perception and the types of information-seeking that occur in early infancy. Hirsh considers, in the preceding section, visual and auditory perception and language as they tend to be related in the development of reading behavior, which usually follows the development of language behavior. The effects of various types of social and environmental deprivations on the ability of the organism to handle visual input are discussed by Mason, and the specific effects occurring in the nervous system of various types of visual sensory deprivation are treated by Valverde and Ruiz-Marcos. Finally, the patterns of development of visual information processing during the first year of life are discussed by Kagan.

In addition to the panel discussion on the relationship between various factors involved in visual information processing and reading, three papers specifically relate various types of reading disabilities to the normal processing of visual information. Ingram deals with the neurologic characteristics of persons exhibiting dyslexic behavior. Children with reading disabilities frequently have various types of disorganized visual perception; some of these disorganizations are discussed by Silver and Hagin. Masland points out the relationship between hemispheric dominance and tendencies toward reversal in visual perception input and the role they may play in reading ability.

The very important relationship between research on problems of visual information processing and reading and the health and educational services available to the general public is outlined by Richardson. Perhaps changes in this interservice area of societal organization can contribute more to improving reading achievement than can changes in other areas of the problem. The conference concludes with a panel discussion among reading specialists. The panelists attempt to relate various presentations to the development and treatment of reading disabilities; they give their opinions of the worth of interdisciplinary and interlevel communication as a means of applying research findings to human problems.

JEANNE S. CHALL

Learning and Not Learning to Read: Current Issues and Trends

LEARNING AND TEACHING TO READ

I would like you to pretend that you are taking a reading test. Figure 1 shows part of a test that is given to high school and college students to see how well and how rapidly they can read silently. They read a story very carefully so that they can answer questions about it. At the end of a minute, they are told to stop reading, circle the word they are then reading, and wait for further instructions. They must then answer questions about the story without referring to it.

My reason for asking you to pretend to take a reading test is to make you feel like a beginner—to have you experience again what it means to learn to read. Figure 2 shows the test as it really looks. The shorthand version of the test illustrates, in an oversimplified manner, that there are two basic requisites in reading: knowledge of the notational system and knowledge of the language. Unless you had learned Pitman shorthand, you could not read the shorthand version of the test (Figure 1), because you did not know the system of notation, although, as you can see from the regular version (Figure 2), you could comprehend the language. But even if you had taken the regular version of the test, you might not have gotten an optimal score, because you did not concentrate, found the

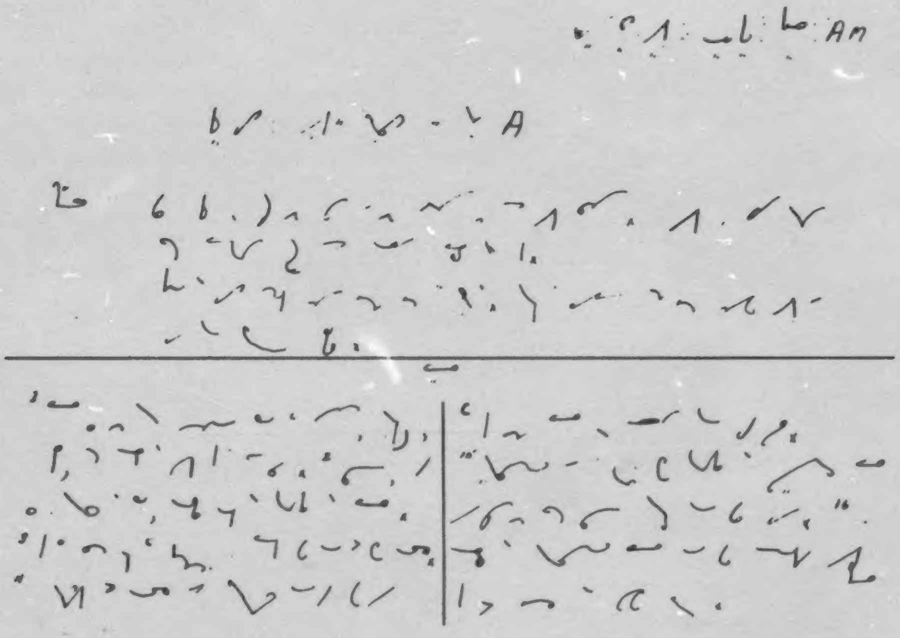
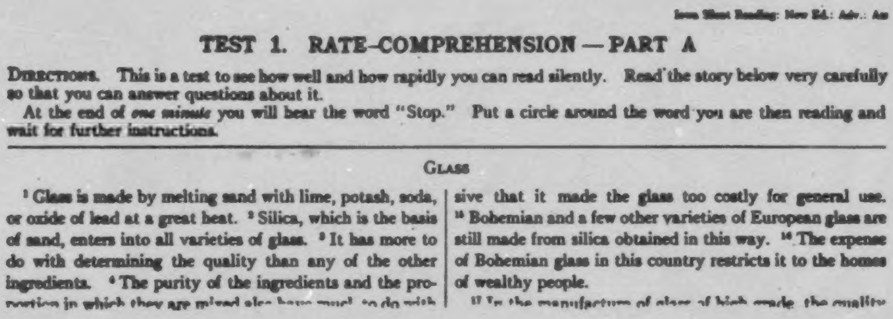


FIGURE 1 A portion of the Iowa Silent Reading Test, transcribed into Pitman shorthand.



Iowa Silent Reading: New Ed.: Adv.: Am

TEST 1. RATE-COMPREHENSION — PART A

DIRECTIONS. This is a test to see how well and how rapidly you can read silently. Read the story below very carefully so that you can answer questions about it.

At the end of *one minute* you will hear the word "Stop." Put a circle around the word you are then reading and wait for further instructions.

GLASS

[1] Glass is made by melting sand with lime, potash, soda, or oxide of lead at a great heat. [2] Silica, which is the basis of sand, enters into all varieties of glass. [3] It has more to do with determining the quality than any of the other ingredients. [4] The purity of the ingredients and the proportion in which they are mixed also have much to do with

sive that it made the glass too costly for general use. [13] Bohemian and a few other varieties of European glass are still made from silica obtained in this way. [14] The expense of Bohemian glass in this country restricts it to the homes of wealthy people.

[17] In the manufacture of glass of high grade, the quality

FIGURE 2 The part of the test shown in Figure 1, in common notation. (From Iowa Silent Reading Test–New Edition. New York: Harcourt, Brace & World, Inc., 1939–1942. Reprinted with permission.)

content dull, and so on. The essential point is that the child who is beginning to learn to read his native language is in much the same position as a person who does not know shorthand but is asked to read it. He has a pretty good command of his language. It is not advanced, but he has an extensive vocabulary, as the various estimates of the number of words known by the average 6-year-old child testify. (A reasonable estimate is about 4,000 words for the average English-speaking 6-year-old in the first grade.[9]) Linguists also tell us that by the age of 6 he has good control over the major grammatical structures of English.

In a sense, the first task facing the child when he learns to read is mastering the notational system (the written code) of the language he already speaks and understands. How can he best learn this written code? Here is where we find much of the confusion and debate about the teaching of reading. All authors of reading programs acknowledge that the ultimate goal of reading instruction is not mastery of the notational system–i.e., saying, sounding out, or decoding printed words–but getting the meaning of the message. But, in their approach to the beginner, some tend to stress the decoding, rather than the meaning, component of reading as the best route to the ultimate goal. The problem in the teaching of reading in the United States, England, and probably other countries with similar writing systems is how to program these two components of reading for the beginner so that he will ultimately be able to comprehend the printed form of his language as efficiently as or more efficiently than the spoken form.

Decoding versus Meaning Emphasis

I analyzed more than 20 beginning-reading programs, including the two reading series used most widely in the United States during 1962–1965 (the duration of the study supported by the Carnegie Corporation[4]) and innovative programs in print or in an experimental stage at the time (1967).[4] From a rather extensive analysis of these programs, I found that they could be placed on a decoding-to-meaning emphasis continuum. At one end were programs that stressed the decoding component at the start; at the other end were programs that stressed the meaning component at the start.

The most widely used reading series during 1962–1965 (indeed, since about the 1930's) was at the meaning end of the continuum. In such reading programs, the child is viewed as a miniature adult, who, from the start, is asked to react to the printed forms of words, phrases, sentences, and stories. Most of the child's practice time (if the teacher follows the manuals that accompany the readers) is devoted to answering questions on the pictures and the content of what is read. The words used in the beginning books are highly controlled and limited to the commonest in the English language. Only a few new words are added in each lesson. In the early 1960's, the total number of different words taught in the five books (three pre-primers, a primer, and a first reader) of the typical basal series for the first grade was about 250–350. The words are selected on a meaning-frequency principle, i.e., words judged to be within the child's understanding and frequent in the language generally. After the child learns to recognize, "as wholes," about 50 of these words, he is taught to analyze words; that is, he is taught which letters or letter combinations stand for which sounds (decoding). But that is secondary to learning to recognize words as wholes and to "reading for meaning."

Most of the innovative programs of the early 1960's, as well as those predominantly in use before the 1930's in the United States, were at the decoding end of the emphasis continuum. Such programs give more attention, at the beginning, to the systematic teaching of the relationship between the spoken and written forms of words. They usually, although not always, teach the child the alphabet before he is taught to recognize words, or while he is being taught to recognize words. Generally, code-emphasis programs view learning to read as a two-stage process: mastery of the alphabetic code and then reading for meaning. Code-emphasis programs vary, and in my classification I included systematic phonics programs, the so-called linguistic approaches of Bloomfield and Fries that limit the early reading vocabulary to regularly spelled words, and schemes that use an initial modified alphabet—for instance, the Initial Teaching Alphabet (ITA)—with a more regular sound-to-symbol correspondence. Although code-emphasis programs put early stress on learning the alphabetic principle, they also have beginners "read for meaning." But in general, compared with meaning-emphasis approaches, the child spends relatively less time at the beginning on "reading for meaning" and more on mastering the alphabetic principle.

The crucial questions here are: Does the beginning emphasis make a difference? Do pupils do better when initiated into reading by a code-emphasis or by a meaning-emphasis approach? Does one or another of these two approaches produce fewer failures? To answer these questions, I reviewed the research conducted from about 1910 to the time of the completion of the first research report to the Carnegie Corporation in 1965.[4] Included in my analysis were classroom experiments comparing the effects of these two basic approaches, laboratory experiments on learning to read, correlational studies on factors related to success in beginning reading, and selected "classic" clinical studies of children who failed or had unusual difficulty in learning to read.

The evidence was not absolutely clear-cut. But it did indicate a trend: the programs that could be classified as having a code emphasis, rather than a meaning emphasis, produced better reading and spelling achievement. The advantages of code-emphasis programs lasted at least through the grades for which there was sufficient evidence—the end of the third grade or the beginning of the fourth. Few researchers followed their children through the fifth and sixth grades, and none followed them through high school. However, on the basis of the evidence through the fourth grade, I hypothesized that the advantages associated with code-emphasis programs would remain longer, if the reading programs in the later grades were sufficiently difficult to challenge the early superior attainment of the children who had been in those programs. Although the clinical studies analyzed did not have the data to confirm or deny that code-emphasis programs produce fewer children with reading difficulties, I was able to conclude that their problems are probably less serious and more amenable to remedy. In other words, although code-emphasis programs are not guaranteed to teach all children to read easily, they tend to lead to fewer serious reading problems.

There was some evidence, too, that the advantages of code-emphasis initial programs were greater among children of lower mental ability, children of low socioeconomic status, and children who are predisposed to having difficulty in learning to read.

The trends evident from the classroom experiments and clinical studies were supported by the laboratory experiments and the correlational studies. Indeed, knowledge of the names (and sounds) of the letters in kindergarten or early in the first grade came out as one of the strongest predictors of success in first-grade reading in studies done as early as the 1930's up through 1965,[5] and also in the most recent U.S. Office of Education (USOE) cooperative first-grade studies completed in 1966.[7]

Probably more classroom experiments comparing the effects of different methods of initiating the beginner into reading have been conducted since 1965 than were conducted up to 1965. What are the results? Do they support or refute the interpretations that I drew from the research up to 1965?

Fortunately, Robert Dykstra, one of the coordinators of the USOE cooperative studies, has summarized the results and made the comparison for us. He drew together specific data from the cooperative research program pertinent to the issue of effectiveness of code-emphasis as opposed to meaning-emphasis programs in initial reading instruction. Following the classification scheme for beginning reading approaches that I devised for the Carnegie study,[4] he categorized conventional basal reading programs as meaning-emphasis, and linguistic and phonics-first basal reading programs as code-emphasis.

After analyzing the studies that were relevant to this issue, he concluded[8]:

> Data from the Cooperative Research Program in First-Grade Reading Instruction tend to support Chall's conclusion that code-emphasis programs produce better over-all primary grade reading and spelling achievement than meaning-emphasis programs. This superiority is especially marked with respect to pronouncing words orally in isolation, spelling words from dictation, and identifying words in isolation on a silent reading test. It is apparent that concentrated teaching of the alphabetic code is associated with improved initial ability to encode and decode words.

Other Factors

Method alone does not account for all differences. Success within all methods is related to characteristics of the pupil, the school, and the teacher. Indeed, larger differences were often found among schools and teachers using similar methods than among those using different methods. Dykstra,[7] in an earlier summary of the USOE studies based on comparisons of beginning reading programs that varied in characteristics other than meaning or code emphasis, concluded that the total instructional setting and the teacher were probably more important for reading achievement than the specific method used.

This conclusion has some support from one of the cooperative first-grade studies directed by Shirley Feldmann and me.[5] We attempted to find out what it is about the teacher that makes a difference in pupil achievement. Detailed weekly observations of teachers who were ostensibly using the same basal reader (meaning-emphasis) program showed considerable variation in implementing it. Generally, we found little correspondence between what the teachers said they did and what they were actually observed to do. When initial readiness characteristics of pupils were controlled, the following factors were related positively to reading achievement at the end of the first grade: overall teacher competence, a thinking approach to learning, providing children with materials of suitable difficulty (neither too easy nor too hard), and a greater emphasis on teaching the relation between sounds and letters (code emphasis).

What can we conclude from the research through 1965 and from the more recent USOE studies? Certainly, that method itself is not a simple matter, or a sufficient condition for achievement. Even without the evidence available from these experiments, it does not take unusual astuteness to observe that teachers vary in competence and skill in implementing any method; that children vary in background, abilities, interests, and receptiveness to different learning tasks; and that schools vary in expectations and facilities. Thus, any reading program, even if carried out exactly as the author prescribes, tends to vary in its effectiveness. It may very well be that a less effective method in the hands of a good teacher may lead to better reading achievement than a more effective one in the hands of a poor teacher. But that does not deny the importance of method.

CRITERIA FOR MEASURING ACHIEVEMENT IN READING

Achievement in reading is usually measured by standardized reading tests. These are group tests with multiple-choice items and time limits. The results are expressed in terms of the age and grade of a normative population and are usually given as grade-level scores or percentiles. By definition, then, about half the pupils who take a test will score above and half below the grade norm (often referred to as the "national norm"). These tests have floors and ceilings, and are designed for narrow grade ranges. The important point about floors and ceilings is that in taking successively more advanced reading tests as he proceeds through the grades, a pupil may, through fortuitous successful filling in of a few blanks, show increments in achievement while, in fact, he is still illiterate. The ceilings may also underestimate the real achievement of the advanced readers in each grade.[3]

Most standardized reading tests measure a conglomerate of skills and abilities that are often hard to separate. The names of the tests may remain the same at different grade levels (e.g., vocabulary, reading comprehension, rate), but they measure different skills and abilities at different stages of development. In the primary grades, for example, a test of reading comprehension is probably a stronger measure of word recognition and decoding skills than of comprehension, because the words and sentence structure in the selections are usually well within the pupil's verbal comprehension. Beginning at about the fourth grade and continuing into high school, a reading-comprehension test measures more of what would be considered "understanding the message." But even here, a low reading-comprehension score may result from a pupil's inability to recognize the words, rather than from his inability to understand the ideas. Thus, the same grade-level score or percentile rank on the same test may mean different things for different individuals and for the same individual at different stages of development.

Although the newer standardized tests tend to have separate subtests for different aspects of reading, they do not solve the problem entirely. It is still difficult, even with different subtests, to disentangle word-recognition and decoding skills from reading comprehension, and reading comprehension from reading rate. It is also difficult to disentangle word recognition from word meaning on tests usually designated as vocabulary or word-meaning tests. It is therefore necessary, especially for pupils who are not performing as expected, to give additional individual tests.

But even for survey purposes, standardized reading tests have a basic limitation that must be kept in mind: because the scores are relative measures, they do not, except by inference, tell us how much of each of the different component reading and language skills has been mastered. Even if we manage to improve the reading ability of all pupils in the United States, the percentage of "poor" readers will remain the same if standardized tests as we know them today are used, inasmuch as poor readers are usually defined as those who score one or more years below age or grade norms. Indeed, I have a strong impression that the tests for the primary grades published in the 1960's are more difficult than those published in the 1940's and 1950's; i.e., they require a greater mastery of the component reading skills for the same grade-level scores. Thus, a 2.0 for a third-grader in 1960 may represent a higher level of skill than a 2.0 for a third-grader in 1940, but both pupils would be classified as poor readers.

What we need are criterion measures, or mastery tests, so that schools, classes, and individuals can be evaluated not only in relation to each other, as they are now, but on the basis of their mastery of known component skills of reading.

DIAGNOSIS AND TREATMENT OF POOR READERS

Current Status

Methods of diagnosis and treatment of poor readers vary a good deal, depending on the facilities and resources available and the viewpoints and skills of those who diagnose and treat. There is also considerable variation in the criteria used to identify poor readers. Generally, in large-scale surveys, pupils who score one or more years below the national norm for their age or grade on a standardized reading test are classified as "retarded" or "poor" readers. This classification therefore includes both children who have limited intelligence and those who are deficient in other areas that may be causally related to reading achievement–children with sensory defects; those who are neurologically damaged, discrepant, or immature; those in the lower socioeconomic levels of the population; those whose emotional problems prevent them from learning; those who have had inadequate instruction; and those with combinations of these characteristics.

The "retarded-reader" classification fails to include those who are exceptionally able intellectually, but who manage to score only "on grade level." Such a classification also overlooks another important distinction: a 1- or 2-year retardation from age or grade norms in the primary grades is different from and probably more serious than such a retardation in high school, where a one- or two-grade retardation may often be a function of a low rate of reading.

There is a growing tendency in schools and clinics to move away from that rather global definition of "poor" or "retarded" readers and to use, instead, the concept of the "disabled reader." The disabled reader is a child who reads one or more years below the norm for his age or grade level and below his mental age. Although this classification eliminates those who have difficulty because of intellectual deficiencies, it, too, poses problems. If group intelligence tests (which usually, except for the first or second grade, require that the questions be read) are used, many "disabled" readers will be overlooked. Indeed, when only school-administered group intelligence and achievement tests are available for estimating the extent of reading disability, it is difficult to tell whether a low reading score can be attributed to a low IQ, or a low IQ to a poor reading score. As with the more global "retarded-reader" classification, exceptionally able pupils who achieve at grade level or above but who are nevertheless still achieving significantly below their potential are not usually classified as "disabled" readers.[11]

Trends

I would like to sketch very briefly some of the broad trends in diagnosis and treatment of poor readers.

As indicated earlier, a good deal depends on who makes the diagnosis and who is responsible for the treatment. In most school systems, the major burden of identification, diagnosis, and treatment still rests with the classroom teacher. Ideally, teachers should use both standardized and informal reading tests to estimate level of functioning, strengths, and weaknesses in component reading and language skills, in order to give each child in the class the appropriate instruction. That is the ideal, but it is extremely difficult to realize. Most classroom teachers are not sufficiently trained in the use of individual tests and in their interpretation, nor do they have the training, the time, or the proper materials to vary instruction on the basis of these interpretations.[1]

More and more schools or school systems (but probably not enough to keep up with the increase in the population of school-age children) have been appointing reading specialists to help the classroom teachers (or the children with reading difficulty). Such a reading specialist usually gives individual tests to determine a child's strengths and weaknesses in reading. The child's record card may be consulted for relevant data on IQ, health, previous achievement in reading and related language skills, etc. The reading specialist may then confer with the classroom teacher who will carry out a more individualized instructional program with the child, or the child may receive "corrective" or "remedial" instruction in reading from the reading specialist several hours a week alone or in a small group. The child may also be referred for a physical examination to rule out the possibility of sensory or other defects, or may be examined by a psychologist or consulting psychiatrist if an emotional or behavioral problem is suspected.

Generally, although the diagnosis and treatment by a reading specialist in a school are more analytic and intensive than those by the classroom teacher, they are usually specific to reading. If the child makes progress in the "corrective" or "remedial" reading sessions, little further diagnosis is undertaken. If progress is not made, the child may be referred for a more intensive clinical diagnosis.

A clinical diagnosis, in contrast with one carried out in a school setting, attempts to get at the underlying "cause(s)" of the problem, as well as at the best possible course of treatment. Ideally, a comprehensive clinical diagnosis involves a social worker, a clinical psychologist, a psychiatrist, a neurologist, a pediatrician, an ophthalmologist, an optometrist, and possibly others, as well as an educational (reading) specialist. Most clinics, however, do not have all these specialists, and the child or the parent may be "referred out" to such specialists. On the basis of the findings of the various specialists, a course of treatment is recommended. It may include parent or child counseling, psychotherapy, perceptual–motor training, remedial instruction in reading, and so on.

Unfortunately, such a multidisciplinary approach to diagnosis and treatment, although accepted as ideal, is still quite rare. And even with a full complement of specialists from different disciplines, it is not easy to make a differential diagnosis. My own experience indicates that a psychologically or psychiatrically oriented clinic tends to find that most children referred to them because of reading disability have emotional problems and usually recommends some form of psychotherapy or counseling in addition to or in place of remedial instruction in reading. In contrast, a neurologically or medically oriented clinic tends to find neurologic or physiologic defects, malfunctions, or immaturities. Some neurologically oriented clinics recommend intensive retraining in reading, writing, and spelling, usually with heavy code-emphasis, for pupils who still have not mastered the fundamental literacy skills. More recently, some medically oriented clinics have been experimenting with direct teaching to the particular deficit in visual and auditory perception that the pupil appears to have.[10]

The differences in diagnostic findings among clinics with different orientations may, of course, be explained by a kind of self-referral by parents and school personnel who identify the child as a reading problem to begin with. It is the parent and the teacher who make the first hypothesis about the possible cause, and then probably refer the child to a clinic that "fits" the hypothesis. I would suggest the possibility that the frequency of some diagnostic findings reported by some clinics reflects the particular viewpoint and interests of the clinic personnel. Reading disability is quite an elephant, and all of us still suffer from varying degrees of blindness when it comes to describing the what, how, and why of it. Indeed, during the 1940's and early 1950's, when the psychologic and psychiatric orientation was dominant, most published reports from psychoeducational clinics found that one of the major causes of reading disability was emotional problems. Since the late 1950's, with the neurologic orientation in the ascendant, more children with reading disability seem to be labeled as neurologically immature, perceptually handicapped, and so on.

Another current trend is prevention of reading failure through early identification and treatment, even before the child is exposed to formal reading instruction in the first grade. Largely through the work of Katrina de Hirsch and her associates,[6] some schools are beginning to test children in kindergarten and are setting up transition classes for "high-risk" children, where their specific lacks in visual–motor coordination, visual and auditory perception, language, and attentional processes are treated in a less pressured atmosphere. Also related is the trend in schools to give corrective and remedial instruction in reading to children beginning as early as the end of the first grade. Until very recently, such children would be considered "not ready." They would usually wait for remedial instruction until they reached the fourth grade or higher, by which time their problems were often intensified.

RESULTS

Lack of Documentation

The next logical question is: How effective are the various approaches to diagnosis and treatment? Unfortunately, there are too few controlled studies or even good clinical reports to state with any degree of confidence that the diagnoses were valid, that they helped in specifying the forms of treatment recommended, that the particular treatment was related theoretically or even clinically to the recommended treatment, or even that the treatment recommended was responsible for the observed improvement in reading. With so many possible causes for the reading disability, it will be quite some time before such data will be available. At any rate, it seems to me that, as with psychotherapy, many different forms of treatment can be and possibly are effective, although it is not always clear what it was about the treatment that made for the effectiveness, or whether the treatment's effectiveness could be said to confirm the validity of the diagnosis. Because all remedial treatments (like all psychotherapies) have in common a concern and care for the child, an acceptance of his difficulty, and a promise of hope to him and his parents, some improvement can be expected from all of them. But if we are going to learn more about the phenomenon of reading disability, we will have to do the kind of clinical and controlled research that will ultimately lead to knowing what leads to what.

The few follow-up studies of pupils who received remedial help (not always specified) in university-affiliated reading clinics seem to indicate that children who receive remedial reading up to the point where they can read well enough to function in school do tend to maintain the gains they make. There is also some evidence that the earlier the identification and remedial treatment, the greater the chance for a successful outcome.[2]

Early Identification

What about the effectiveness of programs of early identification and treatment before the first grade? Here, we must await the evidence. The practice of setting up transition classes for "high-risk" children is still too new to know whether the expense of time and effort is worthwhile in terms of progress made in learning how to read or in the overall adjustment of the child. I would hazard a guess that, even though some of these intervention programs are successful in making the child more "ready" for regular reading instruction in the first grade, his success in the regular first grade and later will depend also on the kind and quality of instruction he receives there.

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DISCUSSION

DR. LINDSLEY: Dr. Chall, you have spoken about the broad aspects of the problem of reading, the criteria for reading, and how we have measured the state of the child's problem, and you have spoken about perceptual factors in reading. You did not make a special point of the matter of learning to attend. For example, children are asked to read the Iowa Silent Reading Test silently and then turn it over and answer the questions. I am sure that we have all had the experience, when we have taken a test, of not expecting the content of the test. There is the difficulty of focusing attention on the test and perceiving the relations that are interwoven in the written language. To what extent has this been proved to be a problem, or to what extent have investigators focused on the matter of learning to attend? We speak of learning to attend and learning to perceive, which seem to me to be very fundamental problems.

DR. CHALL: Attention is definitely an important part of learning to read or of learning anything. In fact, I wonder whether some methods are more successful than others because they help to focus a child's attention better. A method that requires a child to trace a word, or write it, and say it at the same time focuses his attention more than just asking him to say the word he sees. In fact, in the earlier classroom experiments that I analyzed for the Carnegie study and also in the more recent USOE studies, reading programs that incorporated writing with early reading instruction showed benefits over those that did not. Another bit of evidence of the importance of attention comes from a small study that I did in 1965 with colleagues at City College of New York (J. Chall, F. Roswell, L. Alshan, and M. Bloomfield, unpublished) in which first-grade teachers were asked to rate, on a five-point scale, their pupils' ability to pay attention. There was a significant relationship between the teachers' ratings of the pupils' ability to pay attention and achievement in reading. Also, one of the factors that de Hirsch found to be a significant predictor of reading failure among preschool children was hyperactivity or difficulty in attending.

DR. DOTY: I recently read a paper (Amer. J. Orthopsychiat. 38:599-614, 1968) by a Japanese group claiming that there was no dyslexia among a large population of Japanese children, whereas the rates for all European languages are rather similar. If this is a valid bit of work, I think it poses an extremely important point as to the nature of the problem. We might be able to determine the heart of the difficulty from such comparative studies.

DR. KAGAN: I know that study. I am not suggesting that the following fact explains it, but 49% of the teachers in those schools were men.

DR. WADE MARSHALL: That brings up the finding that the ratio of boys to girls in children having developmental dyslexia is 5 or 6 to 1. I wonder whether that is simply because boys are more active and are disciplined more often.

DR. CHALL: The Japanese article interests me very much. The first thing that came to my mind when I read it was that it was based on few cases and on teacher judgment. If it is true that there is no dyslexia among Japanese children, or a smaller percentage than in western countries, then many explanations are possible. One explanation that was already mentioned by Dr. Kagan may have something to do with it: the male teacher.

Another has to do with the Japanese notational system, which is syllabic, although it uses ideographs as well. English and other European languages are basically phonemic; that is, the separate consonant and vowel sounds are represented by separate letters or letter combinations. In the literature on reading, we find in both clinical reports and correlational studies that the sounding and blending of phonemes (for instance, |c|a|t|) are particularly difficult for the severely disabled reader. Orton made a particular point about blending difficulty among his clinical cases. In Japanese, the written characters stand for syllables; that is, there are separate visual notations for *na*, *ne*, *ni*, *no*, and *nu* that do not resemble each other. Therefore, the Japanese child does not need to make the fine distinctions in vowel and consonant sounds that the English-speaking child must make, nor does he have to blend vowels and consonants as does the American or English child.

A third possible explanation is that education in Japan is highly valued and highly competitive. A great deal of time is spent on learning, especially on learning to read.

Still another explanation involves the way reading is taught. I think the Japanese and Chinese teach writing early, simultaneously with reading, and even guide the children's hands in writing. And early writing does seem to be related to early acquisition of reading skills.

DR. BERING: What about the fact that the Japanese and the Chinese read vertically, as opposed to moving from left to right?

DR. CHALL: That might have a bearing on the problem. I understand that those languages can be written horizontally as well.

DR. STAIGER: I might add two other possible reasons for the Japanese-language difference. One is the social pressure brought to bear on the child—his whole family loses face if he does not succeed in school. Another is that the Japanese have two systems of writing: one is phonic, and children are introduced to the phonic system and then graduated to the Chinese ideographs. The two-stage system is possibly something like our ITA and other simplified alphabets. Perhaps there is a relationship there.

One comment on the vertical writing: A Japanese friend of mine said that his biggest problem in reading was moving his eyes up and down and he found that, to be comfortable if he read for any length of time, he had to move his whole head, not just the eye muscles.

DR. INGRAM: According to MacDonald Critchley, only one type of Japanese script is likely to be associated with dyslexia, and that is the one that uses phonic spelling. The writing of the syllable for "rain," which may be a cloud with a few drops of rain coming down out of it, is much less likely to involve dyslexia.

I am very surprised to hear that pressure on Japanese children is more likely to reduce dyslexia than to increase it.

It is possible that children with difficulty in reading and writing have difficulty in maintaining their attention for any length of time, and certainly that has been the experience in the Word Blind Institute in Copenhagen, where it was found that the more severely affected the child is, the shorter the period of remedial instruction may be. They may start with five or six periods of 5 or 10 min at a time during the day. I agree that overactive children—for example, the hyperkinetic children described by Prechtl and Stemmer (Develop. Med. Child Neurol. 4:119–127, 1962)—may have difficulty in sustaining attention, but is it not also possible that, if you ask a child to do something that he finds particularly difficult, it will be very difficult for him to maintain his attention span?

DR. YOUNG: I would like to know at what age Japanese children start to read. Some years ago, I talked to some educators who were a part of a group that studied the Russian school system. They mentioned several times in our discussions that Russian schoolchildren do not wear glasses in the same proportions as American schoolchildren. American schoolchildren do not wear glasses in the same proportions as Japanese schoolchildren. One of the differences between the American and Russian school systems was that we start our children reading around the age of 6, and they start a year later. I am curious about when the Japanese start to read.

DR. SHANKWEILER: Dr. Doty raised the question of a cross comparison of language and reading difficulties. I did not know that the western languages had similar rates of reading disabilities. If that is true, we ought to know about it because western languages differ enormously in the degree in which they have predictable phonic structure; it seems to me that differences or similarities in the rates of reading difficulties in western languages ought to be of concern to us. I don't know what the facts are.

DR. STAIGER: I know the figures from the survey claiming that there is no dyslexia in Japan, but the reason that so little is reported is that, in comparison with our country, where the dyslexia ratio of boys to girls is 5:1, there is a 1 : 1 ratio, so that it looks like less dyslexia. But there is an excess of boys to girls here, compared with Japan.

DR. MEIER: I have some data dealing with studies in schools in which pupils started out in kindergarten with male teachers who had absolutely no sophistication in reading instruction, and their reading readiness was greater than that of all other groups. The very conservative school systems still prove that, with the male teacher who has had no formal reading instruction, schoolboys are proceeding with written material much more rapidly than other groups of boys and doing nearly as well as the girls. In fact, their mean performance is such that there is no significant difference between boys and girls.

DR. LANGE: Our school has had a male teacher with the same boys from the first grade through the third grade. I think they have found that the attitude of the boys toward learning is so much better than that of the girls that their whole curriculum is different, and they feel very successful.

DR. HIRSH: May I use "reading disorders" and "dyslexia" as synonymous, or can I say that a child has a reading disorder because he has dyslexia? I used to call a child who did not speak "aphasic," but I was taught by the neurologist to call it a "language disorder" that may or may not be the result of aphasia.

DR. CHALL: I tend not to use the term "dyslexia." I use the term "reading disability," because it does not assume a definite cause for the disability. By "reading disability," I mean a significant discrepancy between what the child is expected to read, as determined by an individually administered intelligence test, and what the child can read. There are so many definitions of "dyslexia," many of which assume a particular etiology, that I tend not to use it. I tend to think of dyslexia in terms of a reading disability stemming primarily from problems that probably have a neurologic basis but do not necessarily show up on the usual type of neurologic examination. The broader terms, such as "reading disorder" and "reading disabilities," include reading problems stemming from cultural, educational, psychologic, neurologic, or sensory deficits or problems.

DR. SILVER: I would like to make a comment on Dr. Chall's herculean comprehensive survey, touching each area of the problem the way she has and defining what she means very clearly. The question of definition of terms has beset us at every meeting of this nature that I have attended. The Interdisciplinary Committee* worked out a compromise definition. We divided problems of language–let us take reading specifically–into groups. In the first group are children who are retarded in reading, which means that they function lower than their age group. This group may include children with low IQ's and children with peripheral sensory defects. In the second group are children with reading disabilities or reading difficulties, which narrows the field a little bit; these are children who are retarded in reading with respect not only to their age, but also to their intelligence and to their educational opportunities (which brings in the cultural factors). This group would include motivational problems, minimal organic defects, and "specific reading disability," which we use as synonymous with "specific dyslexia." This group includes developmental, or so-called congenital, dyslexia and designates children who are retarded in reading with respect to their age, intelligence, and educational opportunities, who have no evidence of central nervous system structural defects, and whose peripheral sensory apparatus is intact. This would include Critchley's group with developmental dyslexia. I think it is important to speak the same language.

I am also reluctant to use "dyslexia" unless I modify it with the term "specific," because it has too many meanings. In general, it would be synonymous with "reading disability," and "specific dyslexia" synonymous with "specific reading disability."

The Japanese study troubled me. As I recall it, there were two groups: those who were taught by means of a phonic method, and those who were taught by means of an ideographic or essentially visual symbolic method. The children who were taught phonetically did better than the children who were taught ideographically. This would be consistent with what we have found (Silver, Hagin, and Hersh, Amer. J. Orthopsychiat. 37:744-752, 1967), but I questioned the study and I am glad Dr. Ingram has reinforced that question. The study really is a little bit different.

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*Interdisciplinary Committee on Reading Problems, Center for Applied Linguistics, Washington, D.C. 20036.

DR. MEIER: I would like to suggest that the beginning chapter of *The Disabled Reader* (J. Money, Ed., Johns Hopkins Press, 1966) is helpful.

DR. SILVER: I might mention that the initial meeting of the Interdisciplinary Committee was bogged down on just this point of terminology. We tabled the question by handing it to a subcommittee; after a year, we have come out with our definitions.

DR. CHALL: The same thing has happened in the latest committee on dyslexia, the National Advisory Committee,* of which I am a member. We have one task force assigned to definitions, and I think they have, at last count, 14 definitions of dyslexia. It will be helpful if they suggest some guidelines.

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*Secretary's (HEW) National Advisory Committee on Dyslexia and Related Reading Disabilities, Building 31, Room 8A34, National Institutes of Health, Bethesda, Md. 20014.

ROLE OF THE VISUAL SYSTEM: OPTICAL AND OCULOMOTOR, RETINAL, AND CENTRAL NEURAL FACTORS

FRANCIS A. YOUNG

Development of Optical Characteristics for Seeing

Although it is not yet possible to describe completely the development of the optical characteristics of the human eye, it is possible, by relating results of published and unpublished studies with some amount of conjecture, to put together a likely description.

Much of the available information concerning the effects of heredity and environment on the development of the optical characteristics of the eye is based on studies of subhuman primates, primarily chimpanzees and monkeys. However, a recent study of the Eskimo population at Barrow, Alaska, which supports the results of studies of subhuman primates, suggests strongly that the early visual environment and early visual experience play an important role in developing and modifying the optical characteristics of the eye, that the reaction of the eye to its visual environment plays a determining role in the development of the optical characteristics necessary for seeing and reading, that the mechanism of this role should be investigated in all children as they approach the reading age, and that the mechanism cannot be effectively assessed by determining the Snellen visual acuity at 20 ft or at 20 in.

REFRACTIVE CHARACTERISTICS

When a person has 20/20 Snellen acuity, it may be said that he is able to resolve letters that subtend 1 min of visual angle; but little can be said about how he accomplishes this resolution. Hirsch[6] has shown that, if one correlates the refractive error determined by retinoscopy with the Snellen visual acuity, one can demonstrate a considerable correlation (+0.95) in persons who show some degree of myopia. I found a correlation of +0.84 between the same measures.[27] Little or no correlation was found in persons who show emmetropia or some degree of hyperopia, provided the hyperopic persons are not old enough to be also afflicted with presbyopia. For practical purposes, the primary value of the Snellen acuity at either far or near distance would be to demonstrate whether a person has some type of visual refractive error large enough to prevent compensating for it through the use of accommodation, head tilt, squinting, or other techniques.

The refractive characteristics of the eye may be readily determined by retinoscopy, either with or without a cycloplegic drug, such as atropine or Cyclogyl (cyclopentolate). This technique provides an adequate description of the gross refractive characteristics of the eye and indicates whether the person has any degree of astigmatism in conjunction with a measurable degree of myopia or hyperopia. This approach, which requires some clinical skill, permits the clinician to determine which lens or combination of lenses will bring about the neutralization of his retinoscopic shadow in a given eye under a given set of conditions. If the eye is under complete cycloplegia (which may be produced by administration three times daily of 1% atropine for 4 or 5 days), the clinician may state the minimum refractive ability of the subject's eye. Because he is examining under a state of maximally relaxed accommodation, not an ordinary state, he cannot accurately predict what will happen when the eye is functioning without the effect of the cycloplegic. If he performs retinoscopy without the use of a cycloplegic drug, but attempts to induce relaxed accommodation through the use of a plus lens, he can describe more accurately what the subject can accomplish visually under more nearly normal conditions. In that case, the clinician may be deceived effectively by the subject, and conclude that he has achieved a rather basic measure of refractive characteristics, whereas his results may actually be affected by a considerable degree of accommodation within the eye; he is not on much safer ground if he depends on a subjective refraction, calling for the patient's cooperation when he places lens combinations in front of the patient's eye until the level of visual performance is satisfactory.

In any determination of the refractive characteristics of the eye, one can discuss the findings in terms of the types of lenses required to neutralize the movement of the retinoscope shadow or to achieve maximal acuity. If the person requires a minus, diverging lens to neutralize the movement or to achieve maximal acuity, he may be said to have myopia. If he requires a plus, converging lens to accomplish the same ends, he may be said to have hyperopia. If he requires no lens, he may be said to have emmetropia. If he requires a plus or minus cylinder to correct astigmatism, he may be said to have hyperopic or myopic astigmatism. The rest of this discussion will deal with the lens characteristics required to neutralize the movement of the retinoscope shadow or to achieve the best subjective acuity. An eye that requires a minus lens will be a "myopic eye"; no lens, an "emmetropic eye"; and a plus lens, a "hyperopic eye." The distinction between myopia and nonmyopia is arbitrarily taken as the use of a minus lens to neutralize the movement of the retinoscope shadow or to achieve best visual acuity subjectively.

OPTICAL CHARACTERISTICS

The determination of the refractive characteristics of the eye, although a great improvement over the determination of simple visual acuity, does not adequately describe the optical characteristics of the eye. The refractive characteristics depend on the eye taken as a whole. The optical characteristics—the characteristics that contribute to the total refractive capacity of the eye—may change greatly with respect to one another without producing or affecting a refractive error. Thus, an emmetropic person may show a considerable change in axial length, which is compensated by a change in corneal curvature, and still have no refractive error. Retinoscopy or subjective refraction is insensitive to changes in the optical components, unless the changes themselves result in an imbalance of the optical components. Consequently, retinoscopy must be supplemented with some techniques that will provide a more accurate measure of the optical characteristics of the eye. Only by combining such techniques with the measurement of refractive error is it possible to describe what is taking place over time and then to determine the relative importance of changes that occur with growth and with the development of visual ability.

The three techniques most commonly used to supplement retinoscopy and other measures of refractive characteristics are the determination of the corneal curvature by means of keratometry; the measurement of distances within the eye, such as the depth of the anterior chamber, the thickness of the lens, the depth of the vitreous body, and the overall axial length of the eye (by ultrasonography); and the determination of the curvature of the front and rear surfaces of the lens by ophthalmophacometry.

Keratometry permits a highly accurate measurement of the surface curvature of a ball bearing but a somewhat less accurate measurement of the curvature of the cornea, which is not completely uniform but has more than one radius of curvature. However, if the same instrument is used consistently on the same eye, it is possible to develop reasonably accurate measures of changes within the cornea. Ophthalmophacometry is based on the demonstration of Purkinje-Sanson images, or the reflection of light from the surfaces of the cornea and lens. Essentially, it involves directing two points of light into the eye and measuring the separation of their reflections from the back surface of the cornea, the front surface of the lens, and the back surface of the lens (the second, third, and fourth Purkinje-Sanson images).

The combination of measures of refractive error, corneal curvature, depth of anterior chamber, front lens surface curvature, thickness of lens, rear lens surface curvature, depth of vitreous, and total axial length provides an accurate description of the optical characteristics of the eye; anything less than this combination does not. Although we have added considerably to our knowledge of the basic characteristics of the eye, we are still unable to describe exactly what will happen when the eye operates normally, because some of these measurements must be made while the eye is under cycloplegia. Without such a description and accurate measurements from birth onward in the same persons, it is not possible to characterize completely the development of the optical characteristics of the eye. Fortunately, techniques are being developed that will permit the description of ocular performance under dynamic conditions, and it may be hoped that a complete description of the eye, including its static and dynamic characteristics, may be developed within the next decade.

Size

There is no direct evidence dealing with the size of the eye in a living human at birth or during the first 3 years of life. The reasons for the lack of information are related to the difficulty of applying the methods outlined to neonates and very young children. Consequently, most of the information available concerning the overall diameters of the eye has been obtained by measuring eyes post mortem. However, as soon as blood pressure drops, the intraocular pressure drops and the eye becomes quite flaccid; furthermore, most *in vitro* measurements, even when the eye is perfused to reinstate a probably normal intraocular pressure, can vary widely from the measurements that would have been obtained *in vivo.*

Sorsby and Sheridan[17] have provided possibly the best measurements available on the sagittal or anterior–posterior axial length of the eye of the newborn and of children 1–6 days old. There is no significant difference between these two groups, and the mean sagittal diameter is approximately 17.8 mm for both boys and girls. The sagittal diameters are smaller in premature infants, and follow closely the body weight at birth. In the full-term baby of some 3.4 kg, the sagittal diameter is about 17.5–18.5 mm. Therefore, the sagittal diameter increases by around 5–7 mm during growth, if the adult value is taken to be 23–25 mm. It is likely that the usual increase is about 6 mm. There is apparently little growth during the first 2 weeks of life.

Growth

The most complete information available on the growth of the eye in the living human has been provided by Sorsby and co-workers[4,14–18] in a series of studies; they used refraction techniques, photographic ophthalmophacometry, x-ray, and ultrasonic measurements to study the growth and development of the human eye. A series of investigations by van Alphen[21] supplied some of the missing links in our understanding of factors that contribute to changes in ocular size. When the results of these studies are combined with those obtained by me and my co-workers,[23–44] on humans and other primates, a more complete description of the growth characteristics of the optical components is possible.

The sclera, choroid, and retina of the eye of a primate are closely adherent layers with various degrees of elasticity that enclose more or less viscid liquids to form a nearly spherical globe. This globe continues to increase in size after birth. According to van Alphen,[21] the human eye at birth is three fourths of its adult size, and all the ocular structures are probably still growing at birth. The adult size of the cornea is reached between the first and second years, at which time the eye has not yet attained its adult size. Whether the sclera continues to grow after the cornea reaches its adult size is unknown, but intraocular pressure must be important in stretching the sclera.

The adult size of the eye is related to the genetic growth component, the elasticity of the sclera, the intraocular pressure, and a number of other variables yet to be described. In cases of congenital glaucoma, large eyes with large, flat corneas develop as a result of high intraocular pressure and scleral elasticity. In cases of experimentally induced low intraocular pressure, the eye remains small (microphthalmia). But even in these extremes, as well as in most cases between them, the eye remains nearly spherical.

Shape

In the hyperopic eye, a comparison of the transverse, vertical, and axial dimensions based on radiographic measurements of 11 male adult eyes by Deller *et al.*[4] showed no differences between the transverse and vertical dimensions, but did show an axial length significantly longer than either at the 1% level of confidence. In the myopic eye, also, we find that the only significant deviation from sphericity occurs in the axial diameter. Furthermore, if the same significance level is used, there is no difference in diameter between the hyperopic and emmetropic eyes, and the myopic eye is significantly longer than the emmetropic eye in the axial diameter and significantly longer in every diameter than the hyperopic eye.

These comparisons suggest that the eye is normally a sphere and that the shape is determined by the variables of genetics, scleral elasticity, and intraocular pressure. A developing eye that is characterized by a low intraocular pressure and a relatively high scleral rigidity may remain small in diameter even if the genetic component is directed toward greater size. Although one cannot evaluate the genetic component directly, it is possible to measure scleral rigidity and intraocular pressure independently and to estimate the contribution of the genetic component from those measurements.

Sorsby *et al.*[15] conclude that the scanty data in the literature on the dimensions of the eye at birth and in childhood suggest that dimensions almost equal to those of the adult are reached by the age of 2 years. Moreover, it is likely that the cornea has reached its adult size by the end of the first year. Inasmuch as the globe is about 18 mm long at birth and some 5 mm longer by the age of 3 years and there is no drastic change in the refraction of the eye in the first 3 years, compensatory reduction in the powers of the cornea and the lens by as much as 20 diopters must occur during that period.

The development of the myopic eye, with its exaggerated axial length, probably depends on the operation of other variables, although the three mentioned–genetics, scleral elasticity, and intraocular pressure–may also play a contributory role. Sorsby *et al.*[15] also found a second growth period, the juvenile phase, during which the eye grows at a lower but measurable rate to reach its maximum growth at about 13–14 years of age. The hyperopic eye apparently never experiences this later growth, but remains arrested at the infantile phase. The emmetropic eye should also be included in this category, even though it is "compensated," because it does not undergo the later growth changes.

These growth changes are based on changes in axial length, which can be measured with phacometry and ultrasonography. It is not possible to say whether the total size of the eye or only the axial length is increased during the later growth period. Comparisons based on the x-ray measurement of adult eyes suggest strongly that only the anterior–posterior axial length or sagittal diameter increases during this later growth period; it thus causes a change in shape, as well as size, of the globe, which is probably not determined by genetic aspects of the eye itself. The fact that the dimensions of the eye remain relatively stable between the ages of 3 and 11 or 12 supports the concept that new factors contribute to the growth of the eye in the juvenile phase.

MYOPIA AND NEAR-WORK

One of the earliest suggestions as to the nature of the additional variables that may influence eye growth dates back to the early Chinese, who invented lenses and found that the minus lens seemed to assist scholars to see more clearly. In 1813, James Ware[22] presented a paper to the Royal Society of London in which he described his investigation on nearsightedness. He found, for example, that among 10,000 footguards in the British Service not even a half-dozen men were known to be nearsighted. He pursued his inquiry at a military school at Chelsea where there were 1,300 boys; he found that the complaint of nearsightedness had never been made among them until he mentioned it, and even then only three experienced any inconvenience from it. He then inquired at several colleges in Oxford and Cambridge and, although there was great diversity in the number of students who used glasses in the various colleges, glasses were used by a considerable portion of the total number of students in both universities. In one college in Oxford, he accumulated a list of names of no fewer than 32 of 127 students who used either a hand glass or spectacles, between 1803 and 1807. Ware described the effects of fitting concave or minus lenses to nearsighted persons as follows:

> It should be remembered, that, for common purposes, every near sighted eye can see with nearly equal accuracy through two glasses, one of which is one number deeper than the other; and though the sight be in a slight degree more assisted by the deepest of these than by the other, yet on its being first used, the deepest number always occasions an uneasy sensation, as if the eye was strained. If, therefore, the glass that is most concave be at first employed, the eye, in a little time, will be accommodated to it, and then a glass one number deeper may be used with similar advantage to the sight; and if the wish for enjoying the most perfect vision be indulged, this glass may soon be changed for one that is a number still deeper, and so in succession, until at length it will be difficult to obtain a glass sufficiently concave to afford the assistance that the eye requires.

In an appendix to Ware's paper, Sir Charles Blagden[2] gave the following comments.

> Mr. Ware states in his Paper, that near sightedness comes on most frequently at an early age; that it is more common in the higher than in the lower ranks of life; and that particularly at the universities, and various colleges, a large proportion of the students make use of concave glasses. All this is exactly true, and to be accounted for by one single circumstance; namely, the habit of looking at *near* objects. Children born with eyes which are capable of adjusting themselves to the most distant objects, gradually lose that power soon after they begin to read and write; those who are most addicted to study become near sighted more rapidly; and, if no means are used to counteract the habit, their eyes at length lose irrecoverably the faculty of being brought to the adjustment for parallel rays.

The statements appear to be as valid as they were in 1813, and this concept, that the use of the eyes for near-work is responsible for the development of myopia, has a long history in ophthalmology and optometry, but there is inadequate evidence for supporting or rejecting it. Myopia usually develops between 10 and 14 years of age and usually tends to increase with time but to stabilize around 18 years of age. However, there appear to be some persons who do not develop myopia until after age 18; they tend to stabilize around the age of 24. Approximately 8% of U.S. grade school children are myopic, 10–15% of junior high school children, 15–25% of high school children, 25–50% of college students, and 40–60% of graduate students. In a study made at Washington State University, 44% of 400 college freshmen were myopic.[29] By the time this group reached the junior year, 56% had dropped out of the university, and the proportion who were myopic had increased to 50%. Among 148 men in the honors college, 57% were myopic, and among 226 women in the same college, 60% were myopic.

Education versus Intelligence

If this consistent finding–that the proportion of myopic persons increases with years of schooling–is accepted and combined with the known relationship between intelligence level and years of schooling (which parallels that for the development of myopia), it might be concluded that the myopic person who predominates at the higher educational levels is also more intelligent than the nonmyopic. It should even be possible to estimate intelligence by determining the refractive characteristics of the eye. Most of the studies that have attempted to demonstrate a relationship between intelligence and refractive error have found none, except when intelligence was measured by written tests.[7,30] There is a positive relationship between performance on such a test and refractive error: myopic persons tend to score higher than nonmyopic. However, when reading ability is statistically adjusted for, the correlation of refractive error and intelligence approximates zero. The myopic person is a substantially better reader than the nonmyopic.

Myopia and Personality

Studies of the personality characteristics of myopic and nonmyopic persons indicate that there are consistent personality characteristics associated with myopia.[12,20,29] In general, the myopic person tends to be introverted, and the nonmyopic, extroverted. Several investigators have found that myopic persons on the average make significantly better grades in college than emmetropic or hyperopic students, tend to be more introverted in thinking and in social behavior than emmetropes, and are more emotionally inhibited and less inclined to motor activity than nonmyopes.[20,29] It may be asked whether the myopic personality characteristics are present before the myopia develops and lead to the development of myopia, or whether the myopia causes the development of the personality characteristics. Apparently, the personality characteristics precede the development of myopia, inasmuch as the characteristics may be distinguished as early as the kindergarten and first-grade years, whereas the myopia usually does not develop until the fifth- and sixth-grade years.

In spite of the consistent pattern that has been described, it is not possible to conclude without more definite investigations that reading leads to myopia. It is clear that most children learn to read in this culture at approximately 6–7 years of age, whereas myopia ordinarily does not develop before the age of 11 or 12. Similarly, the development of secondary sexual characteristics does not begin until 12–14 years of age. Many have argued that myopia is a delayed hereditary phenomenon that does not develop until puberty. Sorsby *et al.*[15] specifically considered the possible relationship of height, weight, general growth rate, such traits as color of iris, hair, and skin, and puberty to myopia. They concluded that no correlation could be found. There was nothing to suggest a spurt of ocular growth at puberty, nor did variations in the age of onset of menstruation influence ocular growth or ocular coordination. Others had reported similar findings--that there is no relationship between physical characteristics and the development of myopia or between nutritional characteristics and myopia (provided the nutrition is adequate).[24]

Myopia and Heredity

Although there is general agreement that myopia is related to hereditary or environmental factors, there is no agreement as to their relative contributions. The disagreement is due to the inability of investigators to evaluate these contributions experimentally. Only the experimental approach provides control over variables, which is essential to such an evaluation.

An experimental design that permits control over all variables would be desirable, but one that permits control over either the hereditary or the environmental factor is essential. The use of identical twins in the typical co-twin control study holds hereditary factors constant, and complete control of the environment would permit holding environmental variables constant.

With human subjects, it is easier to hold heredity constant through the use of identical twins than it is to control environmental factors. The effects of near-work on the development of myopia could be tested by having one twin engage in little or no near-work while the other twin did a great deal of near-work. This situation could be replicated over a number of pairs of twins, and the amount of near-work and the age at which it is done could be obtained. Unfortunately, this study has not been made, and it is not likely to be made, because the obstacles faced in obtaining enough twins and exercising the necessary degree of control over their behavior are virtually insurmountable.

The identical-twin approach is not the only adequate experimental test of the near-work hypothesis. The converse of the co-twin study may be used. Subjects of different hereditary constitutions may be exposed to the same near-work conditions. If all subjects develop the same amount of myopia at the same rate, heredity can be assumed to play no role in the development of myopia. If none of the subjects develops myopia at a rate different from that shown by a control group not exposed to near-work situations, environment can be assumed to play no role. Finally, if the experimental subjects do not develop myopia at the same rate, but the rate is significantly greater in the near-work than in the control situation, it can be assumed that there is an interaction between heredity and environment. The problems faced in pursuing this type of experimental approach with human subjects are comparable with those confronted in the co-twin control approach. The availability of animals whose visual characteristics are similar to man's makes the second type of approach feasible because of the degree of control that can be exercised over animals.

EXPERIMENTAL FINDINGS

A series of studies has demonstrated that subhuman primates, particularly monkeys and chimpanzees, develop myopia under experimental conditions that restrict visual space to a distance of less than 20 in. from the eye.[25,26,35-38] About three fourths of all adult monkeys placed in the restrictive space show an increase in minus refractive error, and more than half the animals show approximately one-half diopter of myopia or more within 3 months after being placed in it. If young animals (1 or 2 years old) are placed in this situation, it requires a longer period–4–5 months–before any myopic changes are shown, but once they begin, they proceed much more rapidly than in the adult animals. If adolescent animals (2½–4 years old) are placed in this situation, the onset of myopia occurs in 2 or 3 months, and the total degree of myopia developed is greater than in the adult animals and less than in the younger animals. This suggests the possibility that the younger animals–and, by analogy, the younger humans–are able to withstand the stresses and strains of the near-work situation for a longer period than the older animals, but that, once they start to respond, they are capable of making a greater response than the older animals. In all groups of animals, approximately 65–75% show myopic changes under these experimental conditions.

When a group of newly captured rhesus monkeys were examined, only 12 of 600 eyes were found to have any myopia.[34] Among wild and laboratory monkeys, the mean and median refractive errors were found to be significantly more hyperopic in the newly captured rhesus monkeys, the younger monkeys, and wild monkeys in general. More myopia was found among pig-tailed monkeys, older monkeys, and laboratory monkeys. When the monkey population is separated into wild and laboratory animals and these are compared with the Pullman, Washington, population of human subjects (characterized by a high proportion of readers) and the nonreading Washington, D.C., subjects studied by Kempf *et al.*,[9] there is good agreement between the laboratory animals and the reading human population and between the wild animals and the nonreading human population. This suggests a similarity between the effect of the laboratory environment on the refractive characteristics of the monkey eye and the effect of the undefined characteristic that causes an intellectually oriented population to have a higher incidence of myopia than a nonintellectually oriented population.

Steiger[19] and some of his followers have suggested that myopic persons gravitate to professions in which myopia is an aid, rather than a hindrance, and consequently end up in an intellectual profession. Thus, it may be that myopic monkeys are particularly well suited for laboratory work and that they choose this profession. But if that is the case, why are there so few myopes among the wild monkeys destined to become laboratory animals?

The relationship between wild and laboratory monkeys has been investigated more intensively by comparing animals that have been matched for sex and species, because there are sex and species differences in refractive characteristics among monkeys that parallel the sex differences found in humans.[34] When 299 wild monkeys were compared with 323 laboratory monkeys, the wild monkeys were found to be significantly more hyperopic than the laboratory monkeys. When 143 wild monkeys were matched against the same number of laboratory monkeys on the basis of species, age, sex, and weight, the same results were obtained. Furthermore, when 50 inside-cage animals were matched against 50 outside-pen animals on the basis of species, age, sex, weight, diet, time in captivity, and time spent in cage or pen, the caged animals were significantly more myopic than the outside-pen animals. Because any hereditary contributions were confounded by the random factors involved in capture and placement of the monkeys, and the influences of age, sex, and diet were reduced by the matching procedures used, the conclusion that restriction of visual environment has an effect on the refractive characteristics seems to be supported.

Control animals placed in chairs similar to those used in the visually restricted space but without the visual-restriction hoods showed an average change of one eighth of a diopter over a 1-year period, while the experimental animals were showing changes greater than one diopter over the same period.

An attempt was made to evaluate the effect of different levels of illumination on the development of myopia in the visually restricted space.[35] Three groups of animals were kept in the space for 7 months: three animals at a level of illumination of 25 foot-candles (fc), six animals at 4 fc, and four animals at 0.02 fc. The animals at 4 fc developed an average of three fourths of a diopter of myopia, whereas the animals in the other two groups developed one fourth of a diopter of myopia in the same period. This difference is significant at the 1% level and suggests that the level of illumination plays a role in the development of myopia, with lower levels having a greater influence on the development of myopia than higher levels (possibly because of variations in the amount of accommodation exerted), except for the extremely low levels, which had little effect. In other words, the effect of illumination on myopia increases with an increase in illumination up to 4–5 fc. As the level of illumination is further increased, the effect on myopia decreases.

If near-work has an effect on the development of myopia, it should operate with some relationship to the changes that occur when one looks from a far to a near object. The two major changes are in accommodation and in convergence. To investigate the effect of changes in accommodation, a group of monkeys were placed in the near-work situation until they started to show the changes toward myopia[38]; the animals then continued in the situation but were given three drops of 1% atropine each day for 2 months. During that period, the myopia decreased by about one-half diopter and remained constant for the duration of the study. This suggests that accommodation plays a role in the near-work effect. Several studies by clinical investigators have shown that children placed on cycloplegics show little or no change toward myopia while the cycloplegic is in effect.[1,5] Sato[11] found that Japanese children taking a cycloplegic daily showed a regression in the measured amount of myopia of approximately one-half to three-fourths diopter as long as they were kept on the cycloplegic; shortly after they stopped taking it, they showed a further increase in myopia. Current studies on monkeys kept in the visually restricted space show no changes in refractive characteristics until a spasm of accommodation develops. (Ultrasound measurements and corneal measurements are combined with refractive measurements in these studies.) Under these conditions, the eye accommodates and maintains the accommodation for some period without relaxation. If this spasm is maintained for a month or more, it is followed by an increase in axial length, which apparently continues as long as the spasm continues. Sato reduced the spasm of accommodation through the prolonged use of cycloplegics; but as soon as the subjects were taken off cycloplegics, they returned to their near-work environment and again established a spasm of accommodation, which resulted in further increases in myopia.

Studies by van Alphen[21] clearly support the finding that tension on the choroid is increased during the act of accommodation. If accommodation were prolonged, the tension on the choroid against the vitreous body would tend to reduce the blood flow through the choroid and result in ischemia of the choroid and the retina, which depends on the choroid for its blood supply. This prolonged state of lowered blood supply could result in a gradual weakening of the retina, choroid, and sclera and a stretching of the layers at the posterior pole of the eye, which would increase the anterior–posterior axial length. Subjects who do not have this prolonged spasm of accommodation would probably not develop ischemia or the weakening of the eye itself.

All these findings have been derived from studies on animals that can be placed in a situation of controlled visual environment. However, we are concerned primarily with human subjects, and it is important to demonstrate that the optical characteristics of animals are similar to those of humans. Leary and I have made such a comparison between the development of myopia in chimpanzees at Holloman Air Force Base and human subjects studied in London. This comparison shows that the annual rate of change in vertical ocular refraction is the same in humans and chimpanzees, but with more variability in the latter. The only major difference between the human and the chimpanzee in terms of the optical characteristics that accompany myopia is that the vertical corneal power change decreases in the human but increases in the chimpanzee. The changes in lens power, the power of the eye, and the reduced axial length are comparable in chimpanzees and humans, and in general, the course of development of myopia in humans and chimpanzees is the same except for the previously mentioned variability and the changes in vertical corneal power. As a result of this basic similarity, it should be possible to study the influence of various factors, both environmental and genetic, on the development of myopia in chimpanzees and, by extension, on other primates and to generalize the findings to the humans.

If it may be said that the visually restrictive environment leads to myopia in subhuman primates, what effect, if any, does heredity have on the development of myopia in these primates? I found no relationship between offspring and parents in refractive characteristics.[26] Similarly, when intersibling correlations were determined on human subjects, no relationship was found between one sib and the other in refractive characteristics, although there was a significant correlation in height, weight, and IQ.[23] Thus, environment appears to have a more important role than heredity in the development of myopia.

The monkey studies suggest that there is little or no myopia without restriction of visual space and that, if one's visual space is restricted, myopia will develop and will do so more rapidly under low levels of illumination than under quite high levels of illumination.

STUDIES ON ESKIMOS

Recently, we studied the vision of an Eskimo population at Barrow, Alaska. Several investigators[3,8,13] have found that primitive peoples who do not read or engage in substantial near-work have virtually no myopia. Cass states that "myopia is unknown among the pure-blooded adult Eskimo. The majority have negligible refractive errors and a small number have low hypermetropia."[3] Skeller has indicated that in the Angmagssalik Eskimo population the incidence of myopia is not more than 2%.[13] The Eskimos at Barrow appeared to be a desirable population for study, in that three generations were available within the village. Furthermore, the number of children per family is high, with an average of eight. Only the present generation has had required schooling comparable with that in the older states. Thus, there was the possibility of both a genetic study and a study of the effects of schooling and of reading under low illumination on refractive characteristics.

In Barrow, houses are not equipped with light meters, and the city utility charges its customers on the basis of a unit consisting of the consumption of a 40-W bulb per month. Because the winter months are completely without daylight, students must read either under the adequate fluorescent lighting in the school buildings or in the inadequate incandescent light at home. This is a situation somewhat comparable with that of the monkey studies in which the animals were kept in a visually restricted space under low illumination.

The study was done on 508 volunteer family members on whom complete data were obtained. Because volunteer subjects may be biased in favor of visual problems, a follow-up study was made of all the Eskimo schoolchildren in Barrow under 17 years of age. The proportions of various refractive errors are similar in both groups.

Figure 1, based on data from the original group, presents the proportion of persons requiring a minus lens for correction, by age groupings from 6 to 88 years of age. No myopia exists in the oldest generation—those over 50. This is in line with the reports of little or no myopia among Eskimos. In the second generation, from 26 to 50, there is more myopia among the younger persons: fewer than 5% of the 41- to 50-year-olds have myopia, approximately 23% of the 31- to 40-year-olds, and 44% of the 26- to 30-year-olds. Overall, about 21% of the second generation are myopic. An amazing 88% of the 21- to 25-year-olds have myopia, about 58% of the 16- to 20-year-olds, and 52% of the 11- to 15-year-olds, for an overall 62% of the 11- to 25-year-olds. As indicated earlier, myopia usually does not develop until approximately 11–12 years of age; consequently, we would not expect to see a great degree of myopia below this age level. If the 6- to 10-year-olds are included, the overall percentage for the third generation (6–25 years old) is 43%. We rarely find such high proportions of myopia among white subjects living in the United States or Europe.

Figure 2 is a plot of the mean refractive characteristics of the same

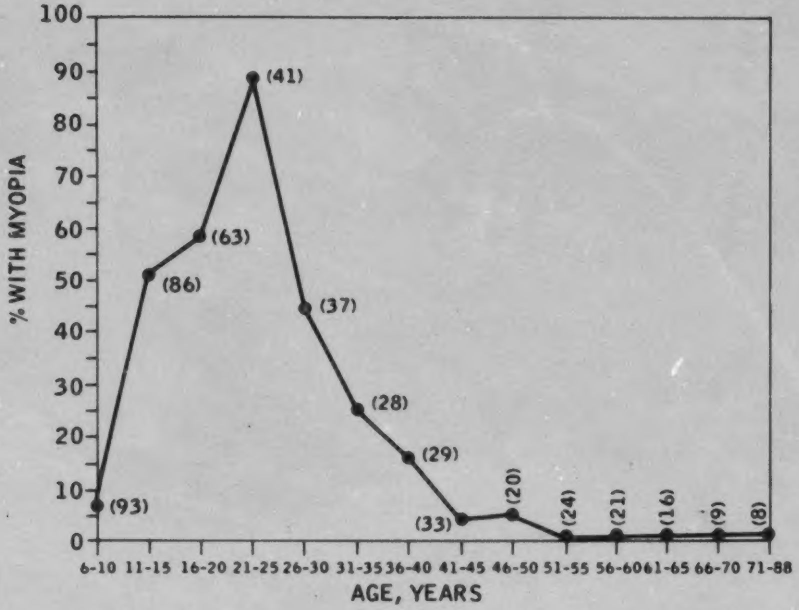

FIGURE 1 **Percentage of myopes in different age groups of Barrow Eskimos (number of persons measured in parentheses).**

age groups. It can be seen again that most of the older persons are hyperopic, but those between 11 and 25 years old are myopic. The average refractive error of the 21- to 25-year-olds is approximately –2 diopters. Most of the members of the oldest generation live the typical Eskimo life, which involves no reading and much outdoor activity, inasmuch as they depend on hunting and fishing to provide their food. At the time of the Second World War, the armed forces began to gather the second-generation men as employees, and these men moved their families into the village of Barrow. These Eskimos learned to read, and the data show that among them the proportion of myopia begins to increase. The persons under 25 years old have had compulsory schooling comparable with that required of children in the older states.

It is difficult to see how such a great increase in myopia could be accomplished by hereditary changes within only three generations. Thus, it appears again that the visual environment plays a large role in the

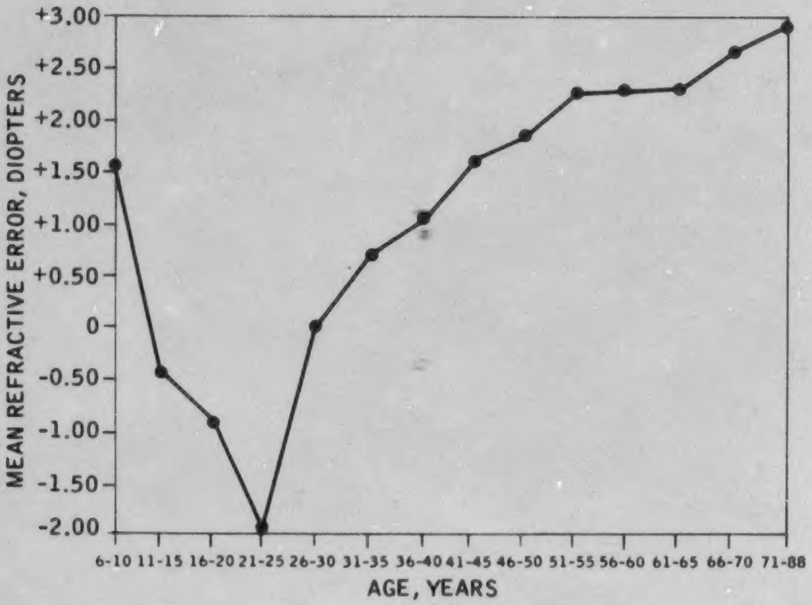

FIGURE 2 Mean refractive error in different age groups of Barrow Eskimos. Same subjects as in Figure 1.

development of myopia and that the suggestions of Ware[22] and Blagden[2] are applicable to this Eskimo population. In addition to reading with poor lighting for long periods, many of these children are overcorrected, which increases the amount of accommodation required for them to read. All these conditions are conducive to the development of myopia if myopia results from an exertion of continuous accommodation.

As has been pointed out by Price,[10] there has been a great change in the diet of the Eskimos during the period in question. That adds another variable that may be related to the development of myopia. Cass[3] explains the absence of myopia among adult Eskimos, and its appearance among children (along with dental caries), as due to change from a traditional high-protein diet to a high-carbohydrate diet. Two facts should be considered in evaluating the possible effects of diet on the development of myopia: the diet is essentially a U.S. diet, but the incidence of myopia far exceeds that in the United States; and diet was not a factor in the development of myopia in monkeys under similar environmental conditions.

OPTICAL CHARACTERISTICS AND READING

Because our purpose here is to determine the role of various factors in the failure to learn to read, we should turn from those who have become efficient readers (and, apparently as a result, have become myopic) to those who do not become efficient readers and usually do not become myopic. As has been stated many times, there are few myopes among school dropouts, and most school dropouts have good distance vision. It is conceivable that this good distance vision may be part of the reason they have never learned to read well. The hyperopic person is capable of seeing quite well at far distance with the exertion of a small amount of accommodation, but must exert an even greater amount of accommodation when he attempts to read. These great amounts of accommodation, which permit him to read, are likely to result in serious visual symptoms that appear to be unrelated to the eye itself. He may develop double vision, inability to concentrate, blurred images, nausea, and general malaise. If the hyperopic person avoids reading, he readily avoids all these symptoms. He will be loath to spend any time in reading, because he finds it distasteful. Without practice, one cannot become a good reader. Therefore, he finds it difficult to master his schoolwork at the level at which reading is essential and may eventually become a dropout. If such a person is examined with a Snellen chart for near vision, he is likely to show normal visual acuity for short periods, and his visual problems are likely to be ignored.

The emmetropic person should be able to read effectively as well as to see clearly at long distance and should not have to exert any more accommodation to read than a corrected myope would have to exert. Thus, he should be as comfortable as the myope in reading. However, the myope apparently can adjust satisfactorily to reading, so much so that he does a great deal of it, whereas the emmetrope may or may not be equipped to become an efficient reader, and whether he can become one can be determined only by the skillful application of visual measuring techniques, and not simply by measuring visual acuity at long or short distances. Because the myope has exerted a self-selection process, in that he has tested himself for reading and has found that he can accomplish this task efficiently, we could probably conclude that the average myope is better equipped to read than the average emmetrope. Furthermore, inasmuch as the myope shows a decrement in visual performance on the Snellen distance acuity chart, he would be referred to a vision specialist–either an ophthalmologist or an optometrist. The specialist would subject him to a clinical examination and determine whether he had any other problems, such as suppressions, anisometropia, or phorias, that could affect his reading performance. Thus, he would have demonstrated a general ability to read and would be properly fitted with glasses to take care of a visual deficiency. The emmetrope, however, would successfully pass the Snellen test, would not be referred to a specialist, and, consequently, could have any number of unrecognized visual problems that would affect his ability to read.

For example, the emmetrope could have astigmatism of a magnitude that would affect his reading performance and yet be able to pass a Snellen test successfully. He could have accommodative problems that would make it difficult for him to get equally clear images in both eyes, so that his accommodation would be in a continual state of activity, seeking to clear different images on each retina. This activity might cause a good deal of asthenopia or visual discomfort and make reading difficult. The emmetrope could become an alternate suppressor because of the difficulty of handling these different images, and he could pass the Snellen test using either eye or both eyes together, and yet be essentially one-eyed as far as reading is concerned, with a resulting decrease in reading efficiency. He could have convergence problems and overconverge or not be able to converge sufficiently. It is possible to have too much or too little convergence for the amount of accommodation exerted. But the interrelationship between accommodation and convergence cannot be detected by a simple test of acuity or a standard clinical refraction. It should be stressed that the hyperope and the myope may have similar problems, but only the myope is likely to be corrected for these problems, because of the ease of detecting myopia in the school situation.

Any child who has reading difficulties should be given a thorough examination by a qualified orthoptist as well as a qualified optometrist or ophthalmologist. The accommodation–convergence relationship should be carefully investigated; all refractive errors should be corrected and deficiencies in accommodation and convergence should be determined. Only with properly coordinated convergence and accommodation and properly corrected refractive errors can a person be visually equipped to read effectively. This type of evaluation ideally involves a refraction with and without cycloplegia, as well as the thorough evaluation of the accommodation–convergence relationship. Such an examination is usually not given by either the optometrist or the ophthalmologist in his ordinary clinical procedures. Because the person who is reading effectively has demonstrated that he does not have serious problems along these lines, only the person with definite reading problems would have to be examined this thoroughly. Until such visual examinations are made, it is not possible to rule out visual defects as contributory factors in reading disabilities.

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DISCUSSION

DR. ALPERN: Did you find a sex difference in myopia in children?

DR. YOUNG: Yes. In my review of early studies, I found reports that girls develop myopia earlier than boys, but girls read much more effectively than boys.

DR. ALPERN: There is an even more impressive sex relationship. Some workers in Ann Arbor have medically examined most of the population of Tecumseh, Michigan; one of the things they looked at was refractive errors (Francis and Epstein, in *International Conference on Comparability in Epidemiological Studies*, Milbank Memorial Fund Quarterly 43, No. 2, 1965). They found that the age of onset of the increase in myopia is impressively related to the onset of puberty. As far as I know, no one has looked at this aspect of the problem.

DR. YOUNG: We have looked at it in monkeys, and there is no relationship between the development of myopia and puberty in these animals. We do not believe that puberty is an essential aspect. Although a child starts to read in school at 6 years of age, he does not really get into substantial amounts of reading until the sixth or seventh grade. Some intensive reading may occur before the sixth grade, but most children are not required to do any substantial amounts of reading until they get to junior high school.

DR. KEOGH: It is possible that the early experiences of boys and girls differ and that girls have been involved in the kind of things in school that would involve them in near-work from an earlier age than boys.

DR. YOUNG: I think that this is really one of the main differences: that girls may be culturally exposed earlier to near-work situations and develop a much better visual performance, whereas boys find it more difficult to develop an effective near-work performance pattern later in life. If children are exposed to near-work situations earlier, they also run the risk of becoming more myopic, inasmuch as the degree of myopia is related to the age of onset. Perhaps reading glasses or drugs could be used to reduce accommodation and control myopia.

DR. LUDLAM: We are involved in the same type of work as Dr. Young. Myopia, as we have discussed it here, is a developmental type of myopia. There is another type that, luckily, is not very common. There may be 2% of myopes born with a very high degree of error, perhaps something like 10 or 20 diopters of myopia, and it is very interesting in that generally it does not worsen. They may be born with 20 diopters of myopia, and they may reach the age of 20 with the same degree of myopia; or they may even experience a decrease, and the myopia may reduce to 8 diopters over the years. And I am talking about good readers.

The type of myope that Dr. Young has been discussing is different. Ordinarily, the schoolchild has normal vision until the age of 7-10 years and then becomes progressively myopic through the school years, sometimes leveling off at the end of high school and sometimes continuing right on through college. I have patients who are engineers and have this myopic condition increasing right up to their 50's. There is still some question, but the relationship holds pretty well, that progressive myopes in general are good readers. These would be the uncomplicated school myopes.

There are also people who read early and do a lot of reading, and still do not develop myopia.

You mention the refractive errors under the effect of a cycloplegic, a drug that paralyzes the muscle inside the eye that operate the lens (the ocular accommodative mechanism). This suggests the finding of R. E. Bannon (Amer. J. Optom.

24:513-568, 1947) that the effect of the drug on refractive error varies. There is no systematic shift in what is called the cycloplegic error from the manifest error. Some people show more change in error in this shift than others, and the usual error from this source is not small. I think we must remember that these effects may result from the use of cycloplegics.

What monkeys do and when they do it does not seem to be completely relevant to humans. These shifts are due to endogenous factors. What solid evidence do you have?

DR. YOUNG: The solid evidence is that I can show the very same effects in monkeys. When you do not put them into the experimental situation, they do not show an increase in myopia. How relevant this is to the problem in humans, I do not know. The basic refractive characteristics of the eye are determined under a condition of relaxed accommodation, because this is a reproducible condition in which the eye is adjusted for vision at a distance of 20 ft or greater. The state of relaxed accommodation is usually obtained in one of two ways: through the use of atropine or some other type of cycloplegic drug, which acts to block the nerve impulses to the ciliary muscle, or through the use of plus lenses, which cause the retinal image of a distant object to appear blurred, which in turn leads to a reflex relaxation of accommodation. The measurement of refractive error or the optical characteristics of the eye under the cycloplegic condition would determine the cycloplegic error, and measurement under the plus lens, the manifest error. If the drug and the plus lens are equally effective, the refractive errors should be the same under both conditions except for errors of measurement.

Bannon found that the errors are not the same under the two conditions, and, although the drug condition usually shows more hyperopia than the plus lens condition, by about 0.5 diopter, this is not always the case. The present concept of the mechanism of accommodation requires that accommodation be relaxed if there is no stimulation of the ciliary muscle. The "relaxed" state of accommodation may not be as great as possible under all conditions; there may be variations in the tonic state of the ciliary muscle. It is generally thought that the use of cycloplegic drugs over such a period as several days to a week will induce the maximal degree of lenticular relaxation.

A recent study of ours (Amer. J. Optom., in press) repeated the Bannon study but used Eskimos as subjects, rather than Caucasians. Those subjects with no refractive errors or with hyperopic errors demonstrated similar effects in both studies. When Bannon's 594 hyperopic eyes are compared with the 513 similar eyes in our study, 69% of the Bannon eyes and 78% of the eyes in our study show an average increase in hyperopia of about 0.5 diopter under the cycloplegic condition. Bannon had no change in 22%, and we had no change in only 10%. Bannon found 9% and we found 12% showing less hyperopia under the cycloplegic condition. These eyes may represent instances of unreliability of the measurement of refractive error; there is no reason to believe that the lens condition would be superior to the drug condition in inducing relaxation of accommodation.

When persons with myopic refractive errors are compared under the two relaxation conditions, major differences are found. The myopic subjects in our study showed the same results as those demonstrated by the hyperopic subjects: 75% had more hyperopia, 15% no change, and 10% less hyperopia. In contrast, among Bannon's myopic eyes, only 38% had more hyperopia, 36% no change, and 26% less hyperopia under the drug condition. The high proportion with less hyperopia under the drug condition is not easy to explain, if one uses the 9% found with hyperopic cases as an estimate of the unreliability of measurement.

DR. SCHUBERT: A well-known specialist has recommended "learning glasses" that would be plus spherical lenses. What do you think about that in connection with what you have stated? Do you know of any cases in which that has been done?

DR. YOUNG: I would like it. It would be a logical conclusion in terms of accommodation in this myopia problem. I know of a number of cases in which clinicians have used bifocal lenses, which are virtually the same thing. There is some confusion in the literature as to whether bifocals have therapeutic effect. I would say that most of the studies so far have not been well controlled. In cooperation with an ophthalmologist, we have been carrying on one study with several hundred children for the last 8 years; the results look very suggestive. If bifocals are properly fitted and if the plus-lens segment is fitted high enough, the myopia will not increase at the same rate as in children who are not fitted with bifocals.

DR. BOYNTON: You mentioned that the accommodative act causes the retina to move forward. This seems inconsistent with the fact that the axial length of the eye, according to your thesis, becomes longer as a result of accommodation.

DR. YOUNG: Your question is how the elongation process can occur, if, in accommodation, the back part of the eye is tightened and moved forward around the vitreous body. Our studies indicate that, if the subject (monkey or human) develops a continuous level of accommodation or spasm, the increase in axial length tends to follow within a variable period, being shorter for more adult animals and longer for young animals. It is our belief that this continuously exerted tension interferes with the nutrition and metabolism of the retina, choroid, and sclera and results in the weakening of these structures, so that they begin to stretch. As they stretch, the space is filled with aqueous fluid, so that the process is more or less continuous as long as the spasm of accommodation continues.

If the newly developed myope were not fitted with glasses for distance, this process should theoretically reach a stabilization point that would leave him fairly well adjusted for his most common nearpoint distance. However, if he is fitted with a distance correction, the process would be restarted, because he would again be trying to overcome the effect of the distance correction while effectively spending most of his time at a nearpoint distance.

We have no reasonable idea as to the basic mechanism that results in the weakening and stretching of the tunics of the eye. Bill (Exp. Eye Res. 5:45-54, 55-57, 1966) has shown that, during the period of ciliary muscle contraction, the movement of the aqueous from the anterior chamber back through the posterior parts of the eye just beneath the sclera is blocked. This path represents one of the important avenues for the drainage of aqueous humor but may also serve some type of a nutritive function in this part of the eye. If that is the case, then perhaps the blocking of the aqueous plays some role in this process, which results in the weakening and stretching of the retina, choroid, and sclera.

DELWYN G. SCHUBERT

Induced Refractive Errors in Human Subjects

This presentation is based on two published studies that Walton and I conducted at the Los Angeles College of Optometry—one on induced myopia[12] and the other on induced astigmatism.[10] A third study, to measure the effect of induced hyperopia, is being planned.

INDUCED MYOPIA AND FAR-POINT PERCEPTION

Many teachers and reading specialists use tachistoscopes, controlled readers, films, and film strips as parts of reading improvement programs. Most teachers on occasion use chalkboards, flannelboards, and flash cards for instructional purposes. Teachers expect children to respond quickly to materials presented by these methods. Rarely, however, do teachers give attention to the sensory or motor skills required for effective far-point achievement.

In clinical refraction,[4] visual acuity is determined by having the subject identify letters of various sizes at optical infinity (20 ft). No precise time limit is applied; thus, the subject has an opportunity to study each letter. Under these conditions, Hirsch[8] has determined the approximate visual acuities for various degrees of myopia. Weymouth[14] and Weston[13] emphasized the importance of considering the time factor in testing acuity. Bartley[2] systematically explored the relationship of time and distance to visual acuity by showing a single fine line randomly in eight different positions. As the distance was increased, the probability of correct response decreased, and as the exposure time was reduced, the probability of correct response decreased.

The purpose of our studies was to determine the effects of artificially induced nearsightedness on far-point tachistoscopic perception.

Twenty-four college seniors were subjects in our study. They were trained observers with corrected or uncorrected 20/20 vision in each eye and were asked to report on words flashed for 1/25 sec from a tachistoscope under constant light intensity at variable exposure distances:

letter size	expressed as Snellen fraction
8.7 mm	20/20
10.9 mm	20/25
17.4 mm	20/40
34.8 mm (approximately the size of chalkboard handwriting)	20/80

Artificial myopia was induced by convex spherical lenses in increments of 0.25 diopter until the subjects began to suffer stress from the refractive error.

The results are shown in Tables 1–3. It is interesting to note that, although all subjects had 20/20 visual acuity, approximately half the 20/20-size words were missed and three fourths of the responses fell below the 80% level of accuracy. Because a short exposure reduces contrast, we were actually measuring the subjects' contrast sensitivity. As shown in Tables 1 and 2, this factor influences the achievement for 20/25 and 20/40 words, but to a lesser extent than for the 20/20 words, because the larger words subtend a greater angle at the nodal point of the eye and thus are more easily resolved.

It is significant that even 0.50 diopter of induced myopia results in a decrement of performance with every letter size. Table 3 shows an even greater percentage loss when myopia is induced by 0.75 diopter. These findings indicate the need for optimal visual acuity in far-point tachistoscopic training. It is also evident that students with maximal visual acuity have an advantage in classroom situations demanding rapid and accurate interpretation of material at a distance. This advantage is also applicable to distance seeing outside the classroom.

TABLE 1 Correct Responses for 10 Exposures (expressed in averages)

Word Size (as Snellen fraction)	Diopters of Induced Myopia						
	Plano	+0.25	+0.50	+0.75	+1.00	+1.25	+1.50
20/20	5.18	–	–	–	–	–	–
20/25	7.58	5.42	4.92	3.17	–	–	–
20/40	9.62	9.25	8.33	6.00	4.46	–	–
20/80	10.00	9.75	9.46	8.96	8.54	7.17	5.50

TABLE 2 Percentage of Responses Falling below 80 Percent Accuracy Level

Word Size (as Snellen fraction)	Diopters of Induced Myopia						
	Plano	+0.25	+0.50	+0.75	+1.00	+1.25	+1.50
20/20	72.7	–	–	–	–	–	–
20/25	50	70.5	75	95.8	–	–	–
20/40	4.2	8.3	20.8	50	62.5	–	–
20/80	0	0	4.2	12.5	12.5	41.6	62.5

TABLE 3 Percentage Loss with +0.75 Diopter of Induced Myopia

Word Size (as Snellen fraction)	Induced Myopia		Difference	% Loss
	Plano	+0.75		
20/25	7.58	3.17	–4.41	58
20/40	9.62	6	–3.62	37.6
20/80	10	8.96	–1.04	10.4

It is apparent that larger letters permit greater accuracy in tachistoscopic training. The smallest letter for far-point training should be 34.8 mm (20/80); smaller letters result in a decrement of performance, even if the subject has 20/20 vision.

SUBJECTIVE EFFECTS OF INDUCED ASTIGMATISM

Is astigmatism detrimental to reading efficiency? Does it produce symptoms? If so, what are they?

Betts[3] found astigmatism associated with many of his cases of severe reading disability and felt that it was one of the causes of the disability. Eames,[6] however, found a greater incidence of astigmatism among good readers than among unselected ones. Many researchers[7,11,15] report an inability to differentiate groups of good and poor readers on the basis of astigmatism. Several specialists have voiced the opinion that severe astigmatism might prove detrimental to efficient reading in individual cases. Romine[9] thought that "it would seem most important to correct any marked degree of astigmatism," and Cleland,[5] sharing that view, stated that "in severe cases of astigmatism it was found to be closely allied with reading failure."

Several of the foregoing studies involved a comparison of the visual characteristics of students who were successful and unsuccessful in reading, and the investigators did not state whether the refractive errors were corrected or uncorrected at the time of testing. If the subjects were fully corrected, they would be emmetropic, and comparisons would therefore have involved reading ability with normal vision. This idiosyncrasy, along with the conflicting opinions of the investigators, piqued our curiosity. If a group of readers were subjected to induced astigmatism, would they experience adverse visual and psychophysiologic effects?

Astigmatism is a refractive condition in which a variation of refractive power exists in the different meridians of the eye. Generally, one meridian exhibits the greatest power and one the least, and these are known as the principal meridians. The cause is almost always a difference in curvature of the refractive surfaces of the ocular media. Most astigmatism is believed to result from unequal curvature of the cornea.[4]

Astigmatism is the most prevalent refractive anomaly. It is classified into the following corneal types:

With the rule (direct)–the curvature of the greatest power lies vertically

Against the rule (inverse or perverse)–the meridian of greatest curvature lies horizontally

Oblique–the meridian of greatest curvature lies between the vertical and horizontal

Bannon and Walsh,[1] in a study of 2,000 patients with refractive problems, found that five sixths of them had astigmatic errors of refraction; of the five sixths, about 40% had astigmatism with the rule, about 25% against the rule, and about 35% oblique.

Cavara, quoted by Borish,[4] calculated the distribution of different degrees of astigmatism (Table 4). It is apparent that the greatest incidence of astigmatism is between 0.50 and 1.00 diopter, and it is most frequently with the rule. Therefore, for our study, we induced 1.00 diopter of astigmatism with the rule while our subjects, 35 seniors from the Los Angeles College of Optometry, from 22 to 47 years old, performed an intelligence test. At the conclusion of the test, each student recorded his own subjective reactions to the induced astigmatism.

As shown in Table 5, well over half the students (63%) experienced

TABLE 4 Distribution of Degrees of Astigmatism[a]

Amount of Astigmatism, Diopters	Distribution, %
0.50	22.94
0.50–1.00	42.44
1.00–1.50	16.18
1.50–2.00	9.21
2.00–3.00	6.39
3.00+	2.84

[a] Derived from Borish.[4] Number of subjects = 5,241.

TABLE 5 Subjective Effects of Induced 1-Diopter With-the-Rule Astigmatism in 35 Subjects

Effect	Reported		Not Reported		No. Reporting Specific Effects
	No.	%	No.	%	
Visual	22	63	13	37	
Blur					19
Diplopia					0
Distortion					9
Psychologic	24	69	11	31	
Weariness					13
Exhaustion					10
Retreat					16
Headaches	24	69	11	31	
Generalized					6
Intraocular					7
Frontal					14
Temporal					4
Unilateral					0
Occipital					2

visual difficulties—blur and distortion, but no diplopia. Of the 35 subjects, 69% reported adverse psychologic effects from the induced astigmatism. The desire to retreat from the test situation appeared with greatest frequency; weariness and exhaustion were also experienced. Headaches were reported by 69% of the students. Frontal headaches were reported most frequently, but there were also reports of intraocular, generalized, temporal, and occipital headaches. No one reported unilateral head pains. Astigmatism is generally believed to be one of the major causes of ocular asthenopia, and that belief was supported in this study.

The fact that children and adults with uncorrected astigmatism frequently confuse similar letters (m and n, o and c) and similar words (flip, flap, flop) can be explained on the basis of the visual blur and distortion encountered. It has been found that the symptoms of blur, distortion, and headache become limiting factors in reading efficiency. A person plagued with these symptoms is markedly handicapped as a student because he cannot sustain concentrated reading for long periods.

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DISCUSSION

DR. YOUNG: Dr. Schubert, did you have a control group who went through this with blank lenses?

DR. SCHUBERT: No, we did not have a control group.

DR. SILVER: How many hours did you allow the subjects taking the test? How much time was allowed for accommodation?

DR. SCHUBERT: That was a weakness in the experiment. The subjects did not have time to adjust to induced astigmatism.

DR. DOTY: I think one of the weaknesses was that they knew precisely what they were going to be subjected to.

DR. SCHUBERT: They were aware of the facts, yes, but they did not know whether their responses were correct or incorrect. They tried their best, and we found out, as mentioned in the results for myopia, that their accuracy level did drop.

DR. INGRAM: If you tell a group of patients or subjects that they have a temperature of 99F, you will find that they have symptoms. If you tell a similar group of patients who have temperatures of 99F that they have normal temperatures, they will have fewer symptoms. I think you must have a control set of blank lenses to use in the same situation. And I think you might well have had someone who took the same test saying that he could not go on because he had a headache–while he was wearing blank lenses.

DR. SCHUBERT: Would the subjects not perceive almost immediately that they were wearing blank lenses?

DR. INGRAM: I think it is very easy to deceive them this way. If they are myopic, they are going to have symptoms; and even if they are not myopic, all you need to do is tell them what symptoms to expect, for instance, that eyestrain is associated with headaches.

DR. ROBINSON: I have a number of patients on whom we put blank lenses. These were children who complained of vision problems, even though no refractive errors were found by cooperating specialists, who said that the lenses would help. Psychologically, the results were apparently very good, in the sense that the headaches disappeared and the reading improved.

The second point I would like to make was already raised, but let us reiterate it. Your subjects were adults who had not become accustomed to this kind of correction, whereas children who have never seen any other way may not be aware of the correction. I am wondering, therefore, whether the results can be carried over from adults to children.

DR. SCHUBERT: Probably not, although I think it gives us some insight, for example, into astigmatism. It appears that persons who see printing in a blurred fashion might develop a headache; I am sure there are headaches that develop under these circumstances. We are aware of the need to improve these studies and can try to improve them in the future. We plan to introduce another group to minus lenses in an attempt to induce hyperopia. At that time, we can use a control group that would be exposed to plain lenses and plus lenses.

DR. YOUNG: I would suggest using, as well, a crossover group, a blind study.

DR. LUDLAM: Yes, a blind study using plus errors. And the experimenters doing the test should not know who has the lenses and what they are, and the subjects should not know anything about the lenses. Then correlate your results, and I believe these observations will be accurate. Perhaps these students from the College of Optometry, who are trained observers, are not the best subjects.

DR. MASLAND: I suggest that you take a group of subjects who have a refractive error and test them immediately after correcting that error, without letting them know. Give them a set of lenses that exactly correct their error. You might take a group of these subjects and give them intelligence tests on the very day that you fit them with bifocals.

DR. ULLMANN: I would like to suggest one more variation, to determine whether women are more responsive than men to the experience of stress that you have introduced by the test procedure. The commandant at West Point once became very much concerned by the number of cadets (it seemed to be exceptionally large) who wore glasses at a football game. He was concerned whether the requirements for admission to West Point should be changed. This raises the question of how much variation is permissible under stress—how much stress is tolerable. It is possible to straighten out this point with two different subject groups to see whether they accept stress in two different ways or react similarly.

DR. SILVER: The discussion has been in terms of similar errors in both eyes. What about children who have a large error in one eye and normal vision in the other eye? What relationship would this have to reading?

DR. SCHUBERT: I do not know the answer to that, but there are vision specialists here who probably can respond to your question.

DAVID G. COGAN / JERRY B. WURSTER

Normal and Abnormal Ocular Movements

Ocular movements are customarily divided, according to the psychosensory stimulus that evokes them, into vestibular, regard, pursuit, and command categories. Their nature and development may be analyzed by objective observation of the normal infant and by study of congenital abnormalities. In this presentation, we will be concerned first with the definitions and neurologic bases of normal ocular movements and then with some of the abnormal conditions of developmental origin.

NORMAL OCULAR MOVEMENTS

Although the subject of ocular movements divides itself into categoric functions, as though each were a separate anatomic entity, all portions of the brain potentially participate in all movements. But the evidence indicates that discrete areas of the brain have a degree of autonomy in effecting movements in response to particular stimuli, and it is convenient from a pedagogic point of view to emphasize this separateness.

Vestibular Movements

Each labyrinth exerts a net tonic innervation tending to turn and rotate the eyes conjugately to the opposite side. The otolith organs are responsible for the static tonus, and the semicircular canals respond to acceleration and deceleration in such a way as to maintain the inertia of the eye position. Thus, acceleration tends to turn the eyes to the side opposite the direction of gross movement of the head, and deceleration tends to turn the eyes toward the same side as the direction of movement of the head. This vestibulogenic movement is normally present at birth, even in the premature infant, and may be elicited simply by rotating the infant in one's arms.

The otolithic influence on the horizontal and vertical displacement of the eyes is normally masked by the voluntary and optic sources of ocular movement, and its influence on rotary displacement, an almost exclusively vestibulogenic function, is difficult to detect grossly. The influence of the semicircular canals is, however, easily seen in the contraversive displacement of the eyes in response to rotation of the head or irrigation of the ears with warm water (conversely, cold water causes a deviation of the eyes to the side that it is applied to).

Normally, the vestibulogenic movement resulting from displacement of the endolymph in the semicircular canals is relatively slow and continually corrected by quick movements bringing the eyes back toward the primary position. The cycles of slow vestibulogenic phase and fast corrective phase constitute a form of jerk nystagmus that can usually be elicited in the full-term neonate. In premature infants, however, the slow phase alone may be present for days or weeks, so that on rotation of the head the eyes maintain a conjugate deviation to one side, instead of developing a nystagmus. This is called a "doll's-head," or "doll's-eye," movement. The fast or corrective phase in man is mediated through the cerebrum and does not depend on the labyrinth. It is served by pathways that are identical with, or closely allied with, the volitional movements. Thus, persistent doll's-head deviations are characteristic of some supranuclear lesions.

Regard Movements

The movements of regard are those elicited by objects attracting one's attention to an eccentric portion of the field. They are movements of attention and, although they are ordinarily evoked by visual stimuli, similar movements may result from auditory stimuli.

The ocular movements to fixate an object of attention are quick and simulate command movements—with which they may, in fact, be identical. Little is known of the efferent pathways for movements of regard, but they are deficient in animals with bifrontal lesions[4] and in human beings with Parkinson's disease.

The infant shows few movements of regard during the first few weeks of life. Objects that alert his attention, as evidenced by a startle reaction or retraction of the lids, are met with a straightforward stare. Oculomotor apraxia is one condition in which regard movements (and pursuit and command movements) are never fully developed.

Pursuit Movements

Ocular movements in following a moving object are called pursuit or optokinetic movements. Unlike movements of regard (or command), they may be slow and they are only partially under voluntary control. Thus, if the entire environment moves relative to the observer, as in looking out of a train window or in an ideal optokinetic test, the eyes are compelled to follow the moving object. When the eyes have reached a comfortable limit of excursion they are brought back toward the primary position by a quick movement, and again follow the moving objects. The repetitive cycles constitute optokinetic nystagmus, and are a convenient test for the integrity of the pursuit movement.

The standard optokinetic drum has black and white stripes that are rotated at a suitable speed in front of the subject's eyes. The afferent arc of the optokinetic response consists of the visual pathways, although those in only one hemisphere are sufficient to produce a response, whereas the efferent arc is mediated through the parietal lobes of the cortex. Further details of the pathways are somewhat obscure, but integrity of neither the frontal nor the occipital lobes is necessary for the optokinetic response.

In the infant, an optokinetic response may be evoked almost immediately after birth, provided the drum's stripes are large enough to be within the discriminative acuity of the neonate and that the moving objects subtend practically the entire visual field.[3,5] Previous reports that the optokinetic response did not develop until a month or more after birth were based on artifacts of testing.

Command Movements

Command movements are those elicited on command. They are often equated with volitional movements. Command movements are probably mediated through the frontal lobe. That is where one would expect the volitional centers to be represented, and experimental stimulation of a discrete area in the posterior portion of the second frontal convolution causes conjugate turning of the eyes. Lesions in this area, however, cause surprisingly little disturbance of volitional control, unless they involve both frontal centers. The inference is that the volitional control of ocular movements has a large measure of bilateral representation.

The infant appears to have poor volitional control of his eye movements at birth and to be unable to turn his eyes at will or in response to an object of regard for the first few weeks of life. With congenital oculomotor apraxia, there is a permanent defect in volitional control of horizontal movements.

ABNORMAL OCULAR MOVEMENTS

The most common examples of abnormal oculomotor development are congenital nystagmus, strabismus, palsies of conjugate gaze, and oculomotor apraxia. Because these do not necessarily have any relationship with one another, they will be described separately.

Congenital Nystagmus

Nystagmus consists of rhythmic oscillations of the eyes. The congenital or developmental variety is almost always horizontal and conjugate (that is, manifest equally in the two eyes) but varies for different directions of gaze. There are three major types of congenital nystagmus; they will be described here only cursorily, because they have been treated in detail elsewhere.[2]

Congenital *sensory* nystagmus consists of predominantly pendular oscillations and is secondary to poor vision that dates from early life. If vision is lost after 4–6 years of age, nystagmus either does not develop or is abortive. The reason for this critical period is unknown, but it has a counterpart in other fields of neurology, such as speech development and somesthetic deprivation. Some authors have assumed that the ocular oscillations serve a useful function by providing a scan that partially compensates for the lack of central vision; but that assumption is contradicted by evidence that arresting the eyes improves vision. The most reasonable explanation for this type of nystagmus is to regard it as an ataxia of eye movements. Macular vision serves the eye muscles as the position sense that other skeletal muscles derive from proprioceptive end-organs. If macular vision fails to develop, the eyes cannot hold fixation and thus develop an ataxic nystagmus. We have called this a sensory type of nystagmus because, in contrast to other types, the primary defect is perceptive. The magnitude or coarseness of the nystagmus will vary not only with the age at which vision is lost, but with the amount of visual loss. When the visual defect is not severe (e.g., Snellen acuity of 20/50 in the better eye), the nystagmus will consist of fine horizontal oscillations; with visual loss to 20/200 or greater, the nystagmic excursions will be progressively grosser. The so-called "searching movements of the blind" are an extreme of this sensory type of nystagmus.

Congenital *motor* nystagmus consists of a jerking nystagmus with a fast component to the right on gaze to the right and fast component to the left on gaze to the left. The neutral point is the intermediate position at which the eyes are approximately stationary and the vision is approximately normal. When, as is often the case, the neutral point is eccentric, the patient stabilizes his eyes by turning his head. Habitual head-turn is thus a common presenting sign of this type of nystagmus. The motor type of congenital nystagmus is of genetic origin and is especially common in males. It simulates a mild paresis of gaze wherein the patient is unable to maintain fixation to either side. Because the horizontal optokinetic response is also characteristically abnormal in these patients, one may postulate a defect, possibly a failure of myelination, in the pathways from the parietal lobes that mediate the optokinetic reflex. Despite this presumed organic basis, the motor type of nystagmus is not characteristically associated with other neurologic abnormalities.

Latent congenital nystagmus is so called because it is elicited by covering one eye. Customarily, the eyes are stationary, but covering either eye evokes a conjugate jerk nystagmus with a fast component toward the side of the covered eye. The etiology of this type of nystagmus is obscure. It may be related to congenital motor nystagmus, inasmuch as it occurs significantly often with this type. It is not related to the function of simultaneous binocular vision; it occurs commonly in patients with strabismus, and simple disruption of binocular vision does not bring it out. It may be related to a phototonic balance between the two eyes, in that progressive darkening of one eye will cause a proportionately coarse nystagmus.

Strabismus

The complex subject of strabismus may be simplified by considering first the two common forms of functional (nonparalytic) strabismus. These are called "alternating strabismus" when the patient uses either eye for fixation and "monocular strabismus" or "concomitant strabismus" when one eye is used for fixation and the other eye habitually deviates. The basis for alternating strabismus is unknown, but it is as though the patient lacked sufficient stimulus for binocular vision. The vision is usually normal and equal in the two eyes. Monocular strabismus, however, is often associated with hyperopia and requires an excessive accommodation for clear distance vision. This in turn produces an excessive stimulation of the correlated convergence mechanism, with consequent turning-in of one eye. The eye with the greater refractive error is almost invariably the one that turns. If, during the early years of life, the deviant eye is not forced to fixate, through patching of the good eye, the vision fails and the eye is said to have amblyopia ex anopsia. The basis for this amblyopia (popularly called "lazy eye") is obscure, but it seems to be age-dependent. Amblyopia occurs characteristically only in the first 6–7 years of life, during which it can be reversed by forced use of the amblyopic eye. After that age, and certainly after the first decade, amblyopia does not develop and, if already present, cannot ordinarily be reversed.

Paralytic strabismus is the other major type. It is an esotropia (inward turn of one eye) when the lateral rectus muscle is paralyzed, an exotropia (outward turn) when the medial rectus is paralyzed, and a hypertropia when one of the vertically acting muscles is paralyzed. Paralytic strabismus does not differ appreciably, as a developmental abnormality, from that acquired later in life, but the diplopia that is so incapacitating in adult-onset cases is not present in infantile or childhood cases. The young patient readily suppresses the false image.

Contrary to popular belief, strabismus itself causes little impairment of a child's visual functions. The absence of binocular vision and stereopsis causes only minor problems in everyday life, and has no bearing on dyslexia. But it is important to prevent amblyopia before the vision in the strabismic eye is irretrievably lost. The cosmetic effect of strabismus is, of course, also a major consideration.

Palsies of Conjugate Gaze

Lesions in the brain stem cause palsies of conjugate gaze, those in the pons affecting horizontal movements and those in the anterior midbrain affecting vertical movements. In addition to these acquired palsies, paralyses of gaze occur occasionally as congenital or developmental abnormalities. Best known is the Möbius syndrome, in which paralysis of conjugate lateral gaze to either side is associated with facial diplegia. Convergence is unaffected and, except for the compensatory head movements and the expressionless facies, the defect causes no functional handicap.

Congenital Oculomotor Apraxia

This is a condition in which, despite full random and vestibulogenic movements, a person is unable to move his eyes efficiently at will or in following a moving object to either side. Vertical movements are unaffected. Because the fast phase of vestibular nystagmus is apparently served by the same neural arc as that of voluntary movements, rotation of such a patient about a vertical axis causes a contraversive deviation of the eyes instead of a nystagmus. To fix an object to either side, the person turns his head instead of his eyes, but the contraversive deviation of the eyes necessitates an overshoot of the head for fixation. The result is a characteristic head thrust unlike that seen with other types of conjugate palsies of gaze (see Figure 1).

Congenital oculomotor apraxia is sometimes familial. It is not typically associated with other neurologic abnormalities. Static visual functions are normal, but children with oculomotor apraxia are invariably slow readers and, despite adequate intelligence, do poorly in school. They have a true oculomotor dyslexia. Although head thrusts become progressively less conspicuous throughout the first decades of life, the defect is never fully outgrown. The person with oculomotor apraxia continues to have some difficulty in rapid voluntary or following movements of his eyes to either side throughout life, and never becomes a facile reader.

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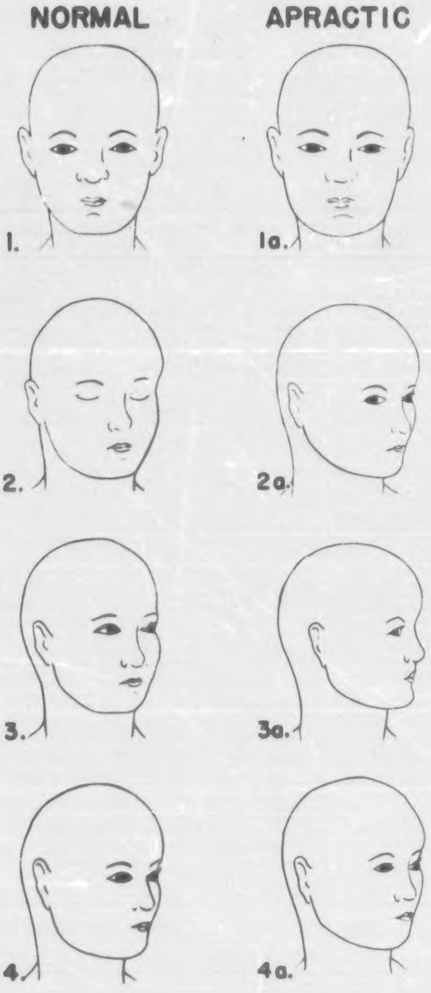

FIGURE 1 Comparison of head–eye movements in the normal and apractic child on gaze to the left. Whereas the eyes precede the head on eccentric gaze in the normal person, the head precedes the eyes in the child with congenital apraxia. In the latter case, the eyes manifest a contraversive deviation, necessitating an overshoot of the head on fixation of a target. (The blink is usually, but not always, present in the normal person and usually, but not always, absent in the apraxic patient.) (Reprinted with permission from Cogan.[1])

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KENNETH R. GAARDER

Eye Movements and Perception

A fundamental topic in understanding reading disability is the physiology of visual information processing. The understanding of that, in turn, depends on a recognition of the role of eye movements in perception. Older work on eye movements in reading (reviewed by Tinker[16]), combined with the ideas I shall present, shows that one way of viewing reading disabilities and perceptual disorders is in terms of ineffective programming of visual input related to faulty functioning of eye movement mechanisms. To understand this fully, it is necessary to grasp the extent to which the perceptual process depends on eye movements. Most of what I shall say is an examination of the mechanisms whereby eye movements mediate perception. Naturally, we will note that eye movements and perception during reading represent only special cases of eye movements and perception in a wider context. By first considering eye movements and perception in general, we hope to be in a position to understand them better in reading and in disordered perception.

I shall attempt to establish two main points: first, that the input of visual information is discontinuous (packaged, sampled, gated, chopped, intermittent, incremental, or step-functioned), with the discontinuities mediated by jumping eye movements; and second, that we may usefully conceive of a hierarchic structuring of intrinsic units of visual perception, wherein eye movements determine the nature of the units at one level. The second point may best be understood by drawing analogies to other information-bearing systems. We shall arbitrarily choose printed language as an example of another information-bearing system, partly because it will lead back to further consideration of one focus of this meeting–reading.

DISCONTINUITY OF VISUAL INPUT

There are two major reasons why scientists have not recognized the discontinuity of visual input before.[9] The first is that vision is subjectively experienced as continuous over time and that our conceptual construct of the real external material world is overwhelmingly one of temporal continuity. As we experience the material world, we are aware of no "breaks" in the time during which our eyes move about, nor does this world seem made up in any way of "pieces." This is in sharp contrast with the facts of the input process. The second reason for not appreciating discontinuity is that, until the arrival of the computer age, the distinctions between "continuous" and "discontinuous" processes were not so concrete as they have been since we have begun to use these problem-solving machines, which are either analog (continuous) or digital (discontinuous). What is at issue is the difference between an information-processing system that takes in information continuously and one that moves in steps, or incrementally, so as to process information in chunks or pieces. A few examples of continuous and discontinuous processes make the distinction clearer. Continuous processes are exemplified by the "coded" groove of a phonograph record and the modulations of radio waves; the discrete, tapped-out letters of a typewriter and the successive frames of a motion picture are discontinuous. Although mathematicians have long been aware of these distinctions, it is only now, with so many of us using computers, that they have become common, experientially understood technologic tools.

Time Course of Saccades

The reason for laboring this point is to bring to your consideration the idea that visual perceptual input is not continuous, as it seems to be, but discontinuous, very much like the successive frames of a motion picture.

But what kind of event in the visual system would represent the changing of the frames? Let us consider some elementary facts about eye movements, overstating to some extent for simplification. The most important fact is that, in moving about to see the environment, the eye moves in virtually only one way—by abrupt, rapid, discrete jumps (exceptions, such as tracking movements, are well discussed in standard texts).[1] Figure 1 shows that we are dealing with a discontinuous process—what engineers refer to as a "step-function" and the technical literature, "saccades." During reading, there are about four of these jumps per second. During other times, while the eyes are open, there are usually at least two jumps per second. The jumps continue during visual fixation, while the eyes fixate a target. The figure shows a typical recording of fixation eye movements showing the size of jump of about 10 min of arc (1/6 of a degree) and a rate of one or two per second.

Before describing evidence of discontinuity of visual information input and processing, I would like to illustrate the manner in which eye movements affect the time course of the perceptual process. Figure 2 shows the effects of gross jumping of the eye about a simple scene. In the upper left is the scene, on which are superimposed five numbered dots connected by lines. The dots represent five successive hypothetical fixations of the fovea (the center of the retina) within the scene, and the lines show the track of the eye over the scene as it jumps. We may assume that the scene was briefly flashed (tachistoscopically) for several seconds on a screen and that during that time the viewer made the five

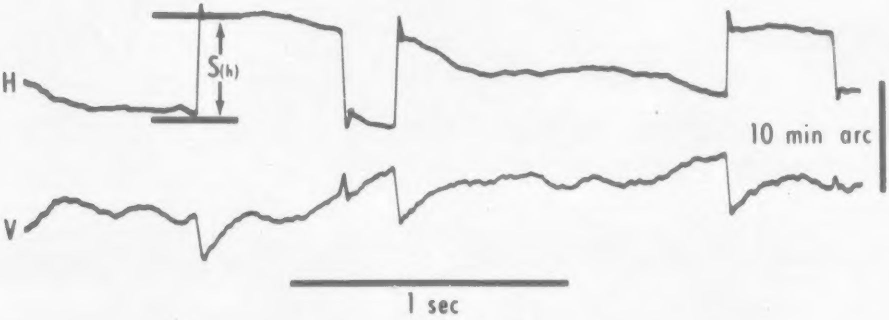


FIGURE 1 Tracings of eye movements during visual fixation, showing several rapid jumping eye movements. H marks the horizontal component of the movement and V the vertical component, recorded by reflecting onto photographic paper beams of light from mirrors mounted on contact lenses.

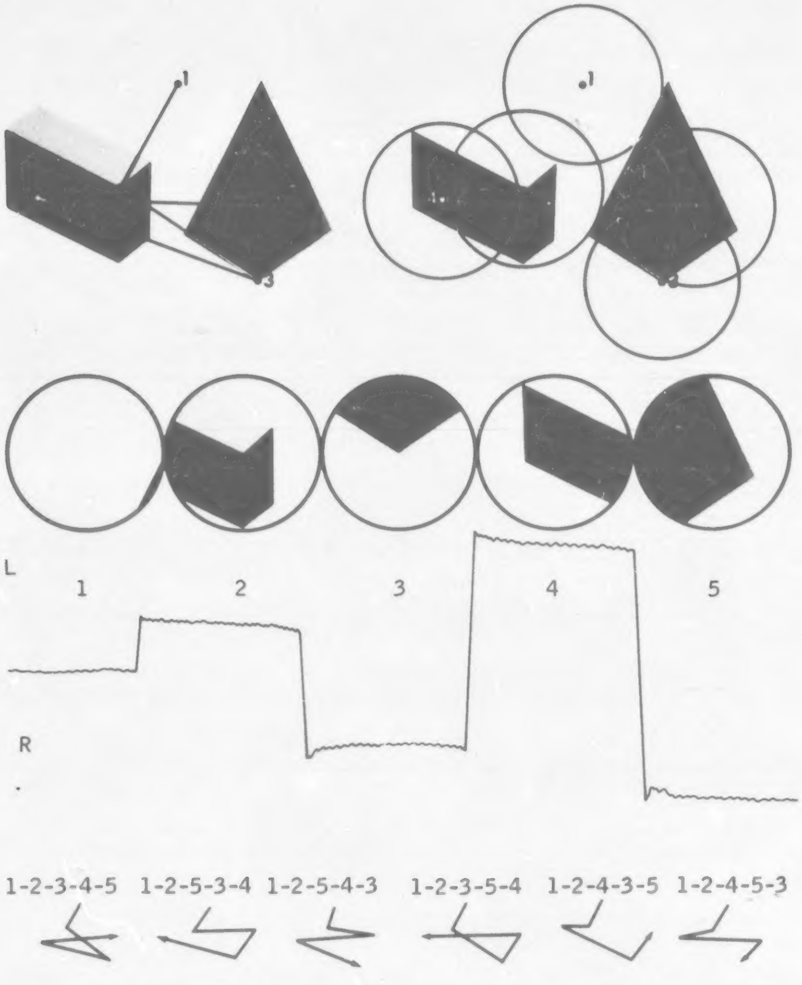


FIGURE 2 Simulation of eye movements in viewing a scene. At upper left is the scene with five numbered dots and connecting lines superimposed. The dots represent five successive fixations and the lines represent the track of the eye jumps between these fixations. The upper right shows arbitrary central retinal areas around each of these fixations. The row of circles simulates the time sequence of presentations to the brain of the chain of five successive central retinal views as the eye views the scene. The tracing below the circles shows the horizontal component of the successive fixations, with L and R representing the left and right directions. The bottom of the figure shows alternative tracks after the first two fixations, illustrating the stochastic nature of the process.

fixations along the indicated track. On the upper right is the same scene with five superimposed circles representing arbitrary equal central retinal areas as they would be on the retina or anywhere back of the retina. Below is a row of circles forming a chain, which represents the sequence in time of these successive central retinal areas as they would be on the retina or anywhere back of the retina. Note that each circle represents a chunk or package of information. The bottom of the figure illustrates two less-important issues to be mentioned in passing. The horizontal tracing simulates a recording of the horizontal component of the eye movements as they occur; at the bottom of the figure, we assume that the same five fixation points were chosen but vary the sequence after fixations 1 and 2.

Edge Visual Images

Figure 3 simulates the effects on the retina of the eye jumps that occur during visual fixation on the same scene. It shows packaging of information, but of a slightly different sort. Because these fixation eye jumps are much smaller than in the previous example, they do not have the effect of causing the same massive transformation of the central retinal area as in the gross viewing eye jumps. Instead, they result in changes at the edges of objects on the retinal image, imitated here by a photographic technique. If positive and negative transparencies of the same scene are superimposed with a slight displacement, the resulting print shows by lightening or darkening of a particular edge the change that would take place on the retina as the result of a small eye jump. The only place where change occurs is at edges. The small arrows represent the vectors (size and direction measures) of the hypothetical jumps that would cause the changes shown. Note that the sets of edges generated are unique to the vector and that the set of vector-generated edges taken as a whole again implies the usefulness of stochastic models. The same thing along a single small arbitrary segment of edge on the retina is shown schematically between A and B in Figure 4 (top). The center of Figure 4 simulates the position of the edge before (t_1) and after (t_2) small jumps of the eye to the left or right, and the bottom shows the net change of these jumps, which would result in "off" or "on" firing of retinal elements, inasmuch as what has happened are "off" or "on" changes.

Figure 5 is a simulation of central retinal areas during reading. The first lines show a sentence of text; the second lines, hypothetical fixa-

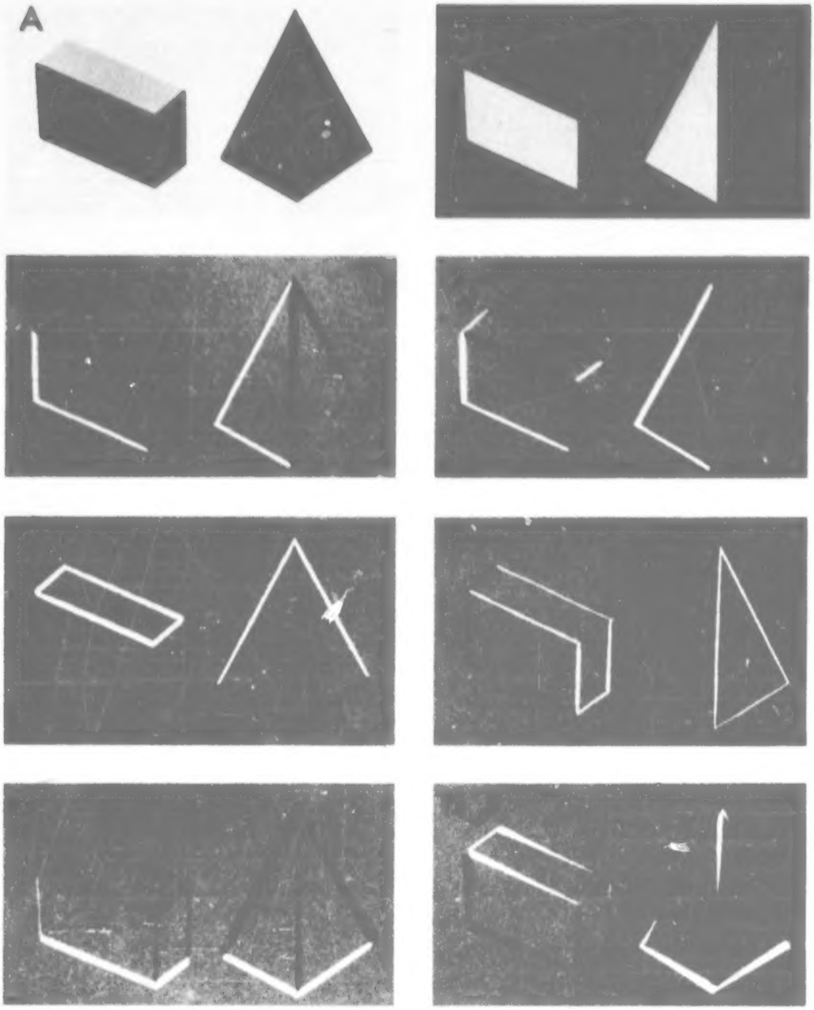


FIGURE 3 Simulation of retinal image edge generation by small fixation eye jumps. This figure shows the positive (A) and negative (B) prints of the same scene at the top. If transparencies of the two are fitted together with slight offset, the discrete edges of C through H result. This simulates the change of the retinal image produced by small eye jumps (indicated by the arrows). For photographic reproducibility, the displacements and the arrows are larger than the jumps that occur during fixation. At 38 cm, 1 deg of arc is about 6 mm. A typical fixation eye jump might result in an apparent displacement of 0.2–1 mm at that distance.

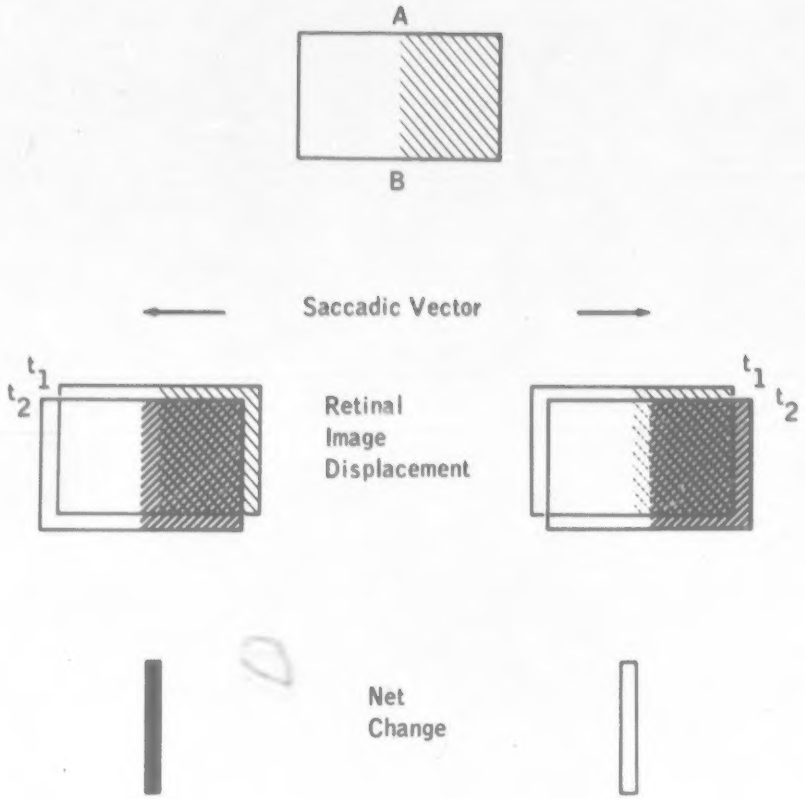
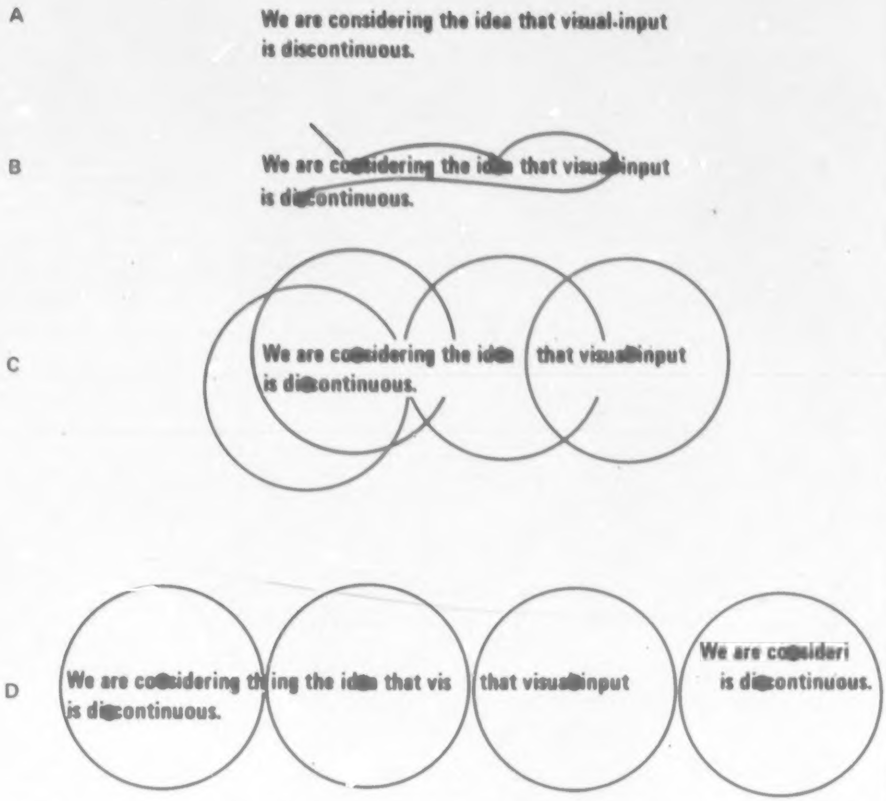


FIGURE 4 Diagrammatic representation of segment of edge on the retina. Top, segment of edge between points A and B (boundary lines are necessary for pictorial purposes). Center, displacement of the edge by a left or right eye-jump vector between time t_1 and time t_2. Bottom, net change of edge produced by the jump.

tions on the text; the third lines, arbitrary central retinal areas around these fixations; and the fourth lines, the time sequence of presentation to the retina and brain of the contents of these successive central retinal areas in a particular chain. It is difficult to accept these processes as representing anything other than discontinuous input mechanisms: each jump of the eye presents the brain with a discrete new package to be processed, and these events occur several times each second.


A
We are considering the idea that visual-input
is discontinuous.
B
We are considering the idea that visual-input
is discontinuous.
C
We are considering the idea that visual-input
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D
We are considering th
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FIGURE 5 Simulation of the effect of eye jumps during reading. A, a short sample of text; B, simulatio of a set of four eye fixations (dots) and the intervening eye-jump tracks (arrows) during reading of the text; C, simulation of arbitrary central retinal areas about each fixation; D, simulation of the time sequenc of presentation to the brain of the chain of four successive central retinal views. Note "overlap" or repetition of words during successive fixations.

Chunking or Packaging of Visual Input

With this picture of the physical facts of the eye-movement system, we can now examine some of the other evidence for and against the idea of visual perception as a discontinuous process mediated by eye jumps. I shall refer to five sets of experiments and their implications.

1. The oldest experimental evidence is the phenomenon of flicker fusion, from which it can be argued that, if, at some particular flicker rate, flicker is not perceived, these chunks of intermittently presented information are subjectively smoothed in the same way as the chunks mediated by eye movements.

2. Conversely, if there were a means to artificially prevent packaging of visual input, it could be predicted that perception would cease, as happens when eye jumps are automatically canceled in stopped-retinal-image experiments.[5,14]

3. Another argument holds that, if perceptual input is intermittent, there must be inhibition of vision during the periods when input is not being processed, i.e., during eye jumps. This is found to be the case during jumps: visual thresholds are raised and inhibitory neurons are activated in the lateral geniculate nucleus.[4,18]

4. Another line of reasoning holds that, if eye jumps establish packages of information, they should be followed by cortical activity marking the arrival of the packages. This is indeed the case: the eye jump triggers occipital activity, recorded as a typical averaged response.[6,11] That the eye jumps are correlated with alpha rhythm is also relevant here, because it shows a relationship between packaging due to eye jumps and more general cortical packaging processes.[10]

5. Less-direct evidence that eye jumps establish discontinuity is provided by the finding of changed fixation eye-jump vectors as a result of changes in visual stimulus. Here, the argument is that, if the form of visual input is controlled by a feedback output of the visual system, changing the stimulus would change the output that controls the input.

Acknowledging these points requires that one conceive of perceptual input as discontinuous, because a discontinuous event (the eye jump) controls it.[7]

Evidence that might be taken to show that eye jumps have no role in perception includes the fact that visual acuity is as good during a flash that is too brief to allow eye movement as during prolonged viewing. One way of interpreting this is to say that if you can see as well during a flash too brief for the eye to jump, then you do not need eye jumps to see.[2] This line of reasoning does not take into account the fact that it is not the eye jump itself that is important, but that the jump causes abrupt incremental change, which is also what the flash causes. In other words, abrupt incremental change of the stimulus is caused by flashing the stimulus or by jumps of the eye and allows vision to occur.

Smoothing of Input

In considering discontinuity of perceptual input, a final point must be made for the sake of logic and completeness: If input is discontinuous, there must be "sampling" periods and "nonsampling" periods.[17] Information that arrives during nonsampling periods must be either lost or held in some sort of short-term buffer memory. Inasmuch as a high percentage of brief light flashes are seen routinely, there must be a short-term buffer memory in the visual system between input (sampling) moments. The direct analogy to time-shared computer technology,[12] which uses short-term buffer memory storage, should be noted.

To recapitulate briefly what has been shown, the input of visual information during perception is not continuous, but is interrupted several times each second by eye jumps, which naturally divide the input into chunks or packages; these packages are reassembled by the brain into a spatiotemporally continuous visual world including, for example, the continuous line of text read from a page; finally, the packages represent natural physiologic units—a step in the direction of reducing phenomena to units whose measurement reflects their intrinsic nature.

ANALOGIES TO WRITTEN LANGUAGE

We shall next explore several neglected areas, with the goal of better grasping some additional aspects of perception without which reading cannot be understood. By developing analogies between visual perception and printed language, we place both the visual system and written language squarely in the generic category of information-bearing systems. We are exploring but several of the issues of interest while passing by others of equal promise (see Polanyi[13]).

The first of these issues is the continuing quest of science for units that are intrinsic to natural phenomena rather than arbitrary. Chemistry and physics made great strides when protons, neutrons, electrons, atoms, and molecules replaced the arbitrary mass–space–time units of grams, meters, and seconds. In neurophysiology, progress is not so easy, but it would be conceded that the nerve spike must represent an aspect of such intrinsic units of nervous activity. Another issue now being perceived is that we are moving about within the domain of information rather than solely within the domain of energy.[3] This means that units of energy and space–time–mass measurement, whether intrinsic or arbitrary, although necessary, are inadequate, and that ultimately perception must be dealt with in informational units instead. A final issue, which also comes from information theory and general systematics, is that our models must be able to encompass the concepts of hierarchy and structure. We will attempt simple definitions of these terms mainly by illustration.

Hierarchy

The three main elements of the analogy to printed language are the hierarchy of levels, intrinsic units, and formation of chains. The first level in the hierarchy of language is a set of letters drawn from the alphabet of that language (Table 1). Each letter is not only an element of the set, but also a natural intrinsic unit of the language, and the units are all of equal size. The next level of a language is a set of words, which make a larger set represented by the dictionary of the language. Words may be considered natural units just as letters are, but they are not of equal size, because they are made up of different numbers of the basic units of letters. The next level of the hierarchy of language is a set of sentences, which may be considered as another type of unit forming a still larger set.

TABLE 1 The Hierarchy of Sets in a Printed Language

Description	Examples
Set of letters (alphabet)	a,b,c,d, . . . ,x,y,z
Set of words ("dictionary")	and, bird, came, doors, top
Set of sentences (ruled by grammar)	Jack rolled the ball. Don't eat mushrooms.
Set of text (ruled by style)	all articles, all books, all manuals

Structure

All these units are combined in various kinds of chains. A given text consists of chains of sentences, which consist of chains of words, which consist of chains of letters. We have seen that all the units in chains are discontinuous, rather than continuous, in the sense considered earlier and, furthermore, that they are units intrinsic to the nature of printed language, rather than arbitrary. Finally, because of the nature of information and the limitations of language, each level of the hierarchy is able to contain only some types of information; the higher one goes in the hierarchy, the greater the degree of complexity that can be conveyed and the more complex the rules for this conveyance. What we are saying is that structure exists, and that it constrains all the possibilities and results in the susceptibility of chains to probabilistic or stochastic models. (A simple example of constraint is that the letters making up a word must be put in the correct order for the word to exist; the structure of the word is a constraint on the set of all possible combinations of the letters in the word, e.g., CAT, CTA, ACT, ATC, TAC, TCA.)

We will now look at how analogy to written language helps us to understand visual perception. The package of visual input mediated by eye jumps is analogous in several respects to the intermediate level of the language hierarchy. They share the properties of being discontinuous, of being intrinsic and natural to the function of the system, of being composed in some way of the smallest units (letters in printed language and nerve spikes in visual perception), and of being made up of various numbers of the smallest units. They also form chains to make up larger units (Figures 2 and 5). It is convenient to consider these packages as analogous to words if we bear in mind that we do not yet know enough about the visual system hierarchy to know whether there are other levels between nerve spikes and eye-jump packages, even though the hypothetically analogous letters and words are on adjacent levels. A great deal more could be said about the ramifications of each of these points of similarity, but, for the sake of clarity, only the three elements mentioned will be established.

Comparison

First, a hierarchy is natural to a language as an information-bearing system; the visual system, as an analogous information-bearing system, must have a hierarchy. In other words, our analogy proposes that complex information-bearing systems are intrinsically and necessarily hierarchically organized. Second, as to the question of units, we have cited two major characteristics of the analogy: the discontinuity of units at all levels (an interesting question is whether it is possible to carry information continuously at higher levels in a system whose lowest level is made up of discontinuous units—the nerve spike and the letter—and so constrains the higher levels to be discontinuous) and the intrinsic natural relationship of the units to the information-bearing system of which they are a part. Third, having noted the formation of chains in both visual perception and printed language, we can sense the importance of eye-jump packages in forming higher units of visual perception—"sentences," so to speak. For example, as you look at an object, the chain of your eye-jump packages will constitute a sentence that is completed when you glance at the next object and begin a new sentence in organizing a percept of it.

We can be specific at two levels as to the nature of the visual system units: the lowest level of vision is a nerve spike, analogous to the lowest level of a written language, the alphabetic letter; and a higher level of visual perception is the package of information (mediated by the eye jump), which is analogous to one of the higher levels of a written language.

THE READING MODEL

We have shown that reading is a process that is divided into its natural units by the jumps of the eye. These units are somehow combined to create both a continuous visual spatiotemporal world and, in reading, a perceptual and cognitive continuity of the textual material. These eye-jump units have a rate of occurrence, with optimal and high and low rates. Thus, there is a framework on which to build a model of reading that involves programming much like that of a computer, with the same kind of vulnerability to faulty microsequences (for example, the various sequences of the letters in CAT) and interference from other sense modalities or cognitive and motor spheres. This model is derived from what we have described earlier, inasmuch as programming is the arrangement of hierarchic units with better and worse alternative sequences and with alternative sets of units from different sense modalities and different spheres, which may or may not be included in the chains. The vulnerabilities referred to can briefly be considered further. The concept of faulty sequences can be amplified by analogy to our present knowledge of computer programming, from which we gain respect for the importance of carrying out a series of operations in exactly the right order. From computer programming, we have learned that, even though there is more than one way to skin a cat, there is a still larger set of ways that will not work at all. In the older literature on eye movements in reading one ineffective microsequence that was studied extensively was the use of regressive eye movements, that is, eye jumps that went back to a part of the text already covered. Another disorder of microsequences involves carrying them out too rapidly or too slowly.[15] Too rapid eye jumps are undoubtedly associated with hyperaroused (overly alerted) states,[8] whereas both too high and too low rates would lead to interference from other spheres.

Our model of visual perception has strong implications for a model of sensory processing and behavior in general that can help us to understand these interferences. It is apparent that, if the visual system is using its own particular coding for its own particular language, each of the other sensory systems is doing likewise, and the same thing is occurring in the cognitive and motor spheres. This can be illustrated most vividly by thinking of ourselves as individual towers of Babel or multilingual United Nations meetings—our eyes might speak German, our ears Arabic, and our stomach French, and the central processor must translate these all into English. If we accept the applicability of these analogies, we are in the useful position of being forced to make choices between time-sharing (i.e., serial processing) models and simultaneous (i.e., parallel processing) models to account for the processing between these different sense modalities. What emerges lucidly for our present concern, however, is the desirability of inhibiting or "turning off" other sense modalities, such as hearing, so as to reduce the interference with carrying out a specific function, such as reading. We can logically and theoretically characterize one class of reading disability as that mediated by interference from other sense modalities or cognitive and motor spheres. (We are not implying that this hypothetical class is uncontaminated by other classes of disability.) Also, it appears that hyperarousal may often characterize this type of disability, but that is another subject.

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DISCUSSION

DR. MASON: You have suggested that there is no way to get from one level to another. That is true of many experiences, and I think that this is the same sort of problem that has stymied psychologists for a long time: How do you get from one level to the next—from the level, say, of letters to words to sentences?

DR. GAARDER: That is the crucial question, and to me it means that you cannot merely know everything about cell physiology, add it up, and make psychology out of it. It will not work. It is a question of enriching pragmatically. It is a question of enriching one level by considering another level, and understanding more about one level by studying it and by referring to higher and lower levels.

DR. BOYNTON: Would you guess that one could establish procedures for the transition from any one level to another?

DR. GAARDER: Yes, in any domain that you care to pick–motor learning, perception, what have you. I think that Dr. Chall had a point about the question of whether children learn written language by coding or phonetics. She suggested that they may need to learn to code before proceeding to the phonetic parts. That may be a very rapid learning process once it is mastered.

DR. LUDLAM: I would like to ask you to do an experiment. Suppose we carefully recorded the series of fixations and saccadic eye movements in the course of someone's reading and then processed them exactly the same way in the same sequence and with precisely the same timing. Suppose, for a second case, that we include the periphery of the visual fields, as well as the central area. I think it is almost certain that we would find that the compensation of the peripherals in artificial conditions would be very poor indeed, and it might be worthwhile to ask why.

DR. GAARDER: The difference between those two situations is that an eye movement that is involved in the first case is absent in the second. The eye movement is under the control of the subject; what the eye is going to do next in the reading situation or in the more general perceptual experience is determined during the 100 msec or more of the fixational pause. I think this is exceedingly important as a problem in visual perception. In the normal situation, clearly, the "computer" knows where the eyes are going to go next and, as a consequence, is able to get a good deal more out of the visual input from successive fixations than could otherwise be possible.

DR. ALPERN: Does this add to the relevance of eye movements for reading?

DR. GAARDER: Yes, I think what the eye is doing in the previous 100 msec is very important in the problem of poor reading, and it is an aspect of the problem that people have not paid very much attention to.

DR. HIRSH: It seems to me you are causing yourself a great deal of difficulty by trying to extrapolate movements of the eye from the stimulus pattern alone. I think there are some rather special differences between eye movements during reading and eye movements around arbitrarily depicted shapes of the type that you show.

DR. GAARDER: I suppose that one of the ways to predict eye movements involves an analogy to the way the language is structured and how well one knows that structure. At least, that appears to be the case.

ROBERT M. BOYNTON

Retinal Contrast Mechanisms

The verb "contrast," according to Webster's New Collegiate Dictionary, means "to exhibit noticeable differences when compared or set side by side." Implicit is the idea that small differences will become less noticeable or unnoticeable if the items being compared are separated. That is true for human vision.[15]

If two half-circles of light, each homogeneous, are very carefully butted against one another, they form a bipartite field. If their luminances are equal, they will form a homogeneous disk without a discernible border between the two half-fields that make up the disk. Suppose that we can adjust the luminance of one half-field independently of the other. The luminance difference necessary for a border to be just perceived is about 0.5%.* But if the fields are moved barely apart–just

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*This value obtains for optimal conditions of viewing, when the just-noticeable difference is based on the standard deviation of many settings, and the subject attempts again and again to set the two half-fields exactly equal in brightness. Here and in experiments to be reviewed later, different conditions and experimental methods will inevitably yield different results. Furthermore, there are significant differences among normal subjects, and in pathologic cases the values obtained may be different by more than one order of magnitude. It is not possible to introduce qualifying statements everywhere in this paper; the reader should accept values given as representative of typical subjects under optimal viewing conditions, unless otherwise stated.

"Contrast" is commonly used to specify a physically measurable difference, as well as to enough to introduce a very thin black line between them—this value will increase to about 1%. Further separation will increase the value even more. Why is this? The answer is much more complicated than might be expected. Indeed, the results of even this simple experiment cannot be fully explained on the basis of our present knowledge, although a general understanding is possible. One purpose of this presentation is to review some of what is known about the retinal contrast mechanisms that underlie this and other observations. Another purpose is to relate this knowledge to the perception of small dark details against a brighter background, which is characteristic of the typical reading task.

RETINAL-IMAGE CONTRAST: GRADIENTS

We must begin by considering what sort of image is formed by an external stimulus on the retina of the eye—the retinal image is by no means a perfect replica of what is outside. In any image-forming system, the image of a point is not a point, but rather an optical-spread function (see Figure 1). Diffraction provides an ultimate limit in any optical system. In the eye, aberration, light scatter, and accommodative errors broaden the function further. Its width also depends on pupil size, being minimal (and thus best) when the pupil is about 2.5–3 mm in diameter. The spread function does not depend on light intensity. At very low light levels, the function describes the probability of arrival of photons, at each spatial position, within a test period.

Assuming the point-spread function as measured by Westheimer and Campbell,[21] an edge between a bright field and one that is completely

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describe subjective experience. In this presentation, "contrast" is used to describe what can be measured with a photometer, and modifiers will be used to refer to the effects produced—optical, physiologic, or subjective—by the physical contrast stimulus.

For small details seen against a large background, contrast will be defined as $(B_t - B_b)/(B_b)$, after Blackwell.[3] Here, B_t is the luminance of the small detail, or target; B_b is the luminance of the background. Here the limit of negative contrast is 1.00 (100%), and positive physical contrasts may assume any value. The justification for this specification is that the visibilities of dark targets (negative physical contrast) and bright targets (positive physical contrast), when seen against the same background, are approximately equal.

In other situations, particularly when bipartite or striped fields are used, there can be no clear distinction between test and background, nor between positive and negative physical contrast. Therefore, physical contrast in these cases is defined as $(B_1 - B_2)/(B_1 + B_2)$. Here B_1 is the positive physical contrast and B_2 the negative physical contrast; the limit of physical contrast is between zero and ±100%.

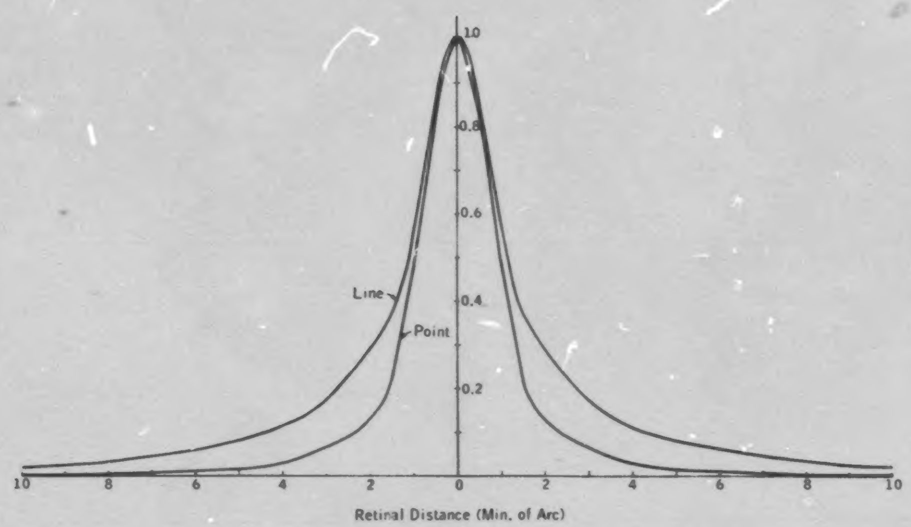

FIGURE 1 Line- and point-spread functions on the human retina, as determined from the experimental data of Westheimer and Campbell[21] by direct physical measurement on the human eye. (Courtesy of G. Westheimer.)

dark produces on the retina the light distribution shown in Figure 2. We conclude that there is a gradient of intensity between the two fields, rather than an abrupt change. It is this sort of gradual variation in illumination that the retina works with, and never the abrupt changes that are so easy to provide outside the eye.*

RETINAL-IMAGE CONTRAST: BLACK LINES

Let us now analyze what happens if the eye is confronted with a dark line, seen against a homogeneous white background. Suppose that the

*For linear and homogeneous systems, a line-spread function such as that shown in Figure 2 can be converted into a modulation-transfer function. The latter shows the percentage of contrast transferred through an optical system, plotted as a function of the frequency of sinusoids of spatial luminance variation. Although this procedure has some advantages, and there have been many experiments in which the response of the eye was examined with spatial sinusoids as the stimulus, the approach is difficult to apply when considering letters of print on a page. For that reason, I do not use the modulation-transfer approach in this discussion.

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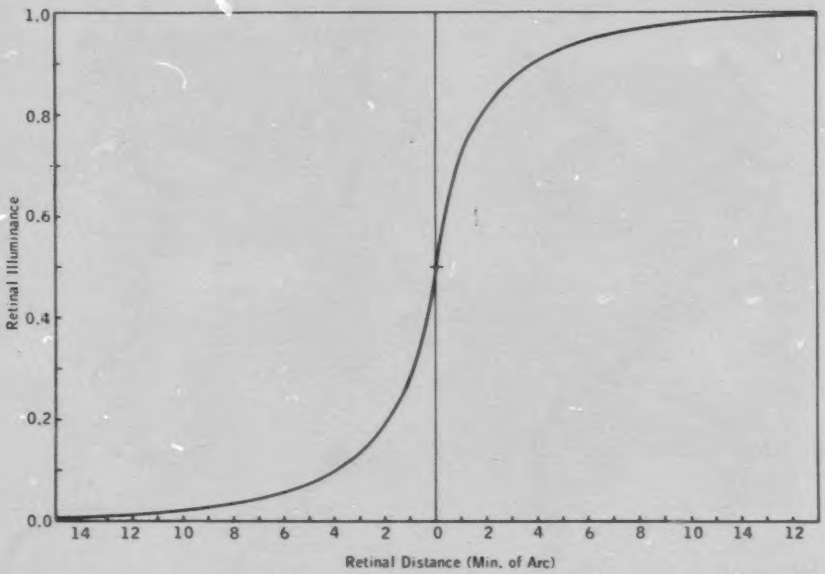

FIGURE 2 Light distribution in the retinal image of an edge in the human eye, best focus, 6-mm pupil, based on the measurements of Westheimer and Campbell.[21]

line is completely black. We can produce this, in the laboratory, by holding a pair of white surfaces–say, 3 × 5-in. filing cards–in front of some kind of light trap, illuminating them diffusely from the front. If the edges of the cards are very sharp and precisely parallel, a thin black line can be produced by bringing the cards very close together. What will the retinal image look like in this situation?

We can find the answer by adding together the two edge gradients, discussed earlier, as shown in Figure 3. Here it will be seen that the closer together the cards and therefore the narrower the line, the lower the contrast on the retina between the illumination at the center of the retinal image of the line and that of the uniform areas flanking it. The retinal contrast produced by lines of various widths is shown in Figure 4.

We can now see that, when we are concerned with the vision of fine lines of high physical contrast, we are, nevertheless, dealing with low retinal contrast. The same is true for more complex forms, such as letters on a page, that are built up from fine lines.

There are two basic ways to increase the retinal contrast produced by looking at a line target. The first is to increase the angle subtended by the line at the eye. This can be accomplished by making the line wider, or by moving a line of fixed width closer to the eye. Increasing the width of the line produces an effect that is easily seen in the use of boldface type. Boldface looks blacker because retinal contrast is higher, although the objective contrast is the same as for regular type (produced with the same ink). The second way to increase retinal contrast is to increase the inherent contrast of the line with respect to its background. This can be done by making the page whiter or the ink blacker. But most inks are black enough so that, even if they could be caused to have zero reflectance, the contrast gain would be rather small. A much greater gain can be had by making the line wider. Nevertheless, the inherently low contrast of a cheap paperback book–caused by small type, low-reflectance paper, and poor control of the width and reflectance of the letters–will result in a noticeable loss of retinal contrast and consequent

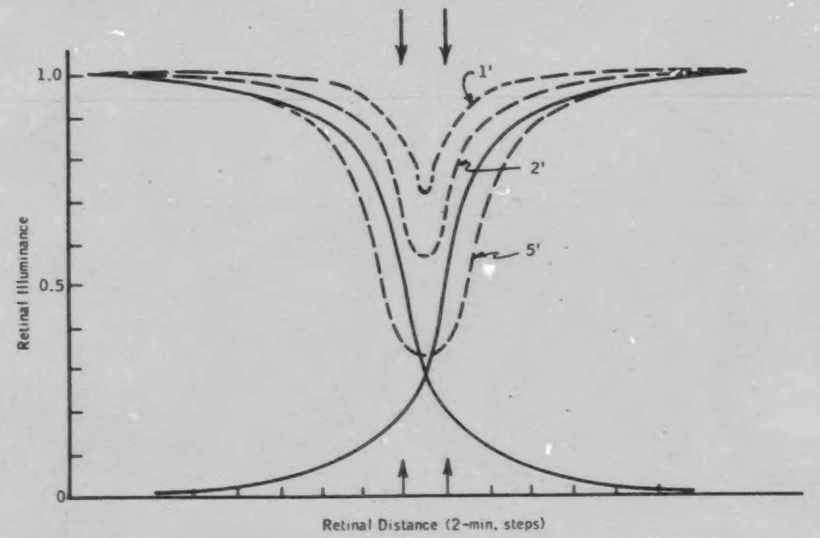

FIGURE 3 Retinal illuminance produced by completely black lines of the widths indicated, seen against a bright background (dotted curves). The curve for a 2-min line has been derived by adding the distributions of the two edges shown by the solid line. The other dotted curves were similarly constructed by moving the edge gradients closer together or farther apart than the distance shown.

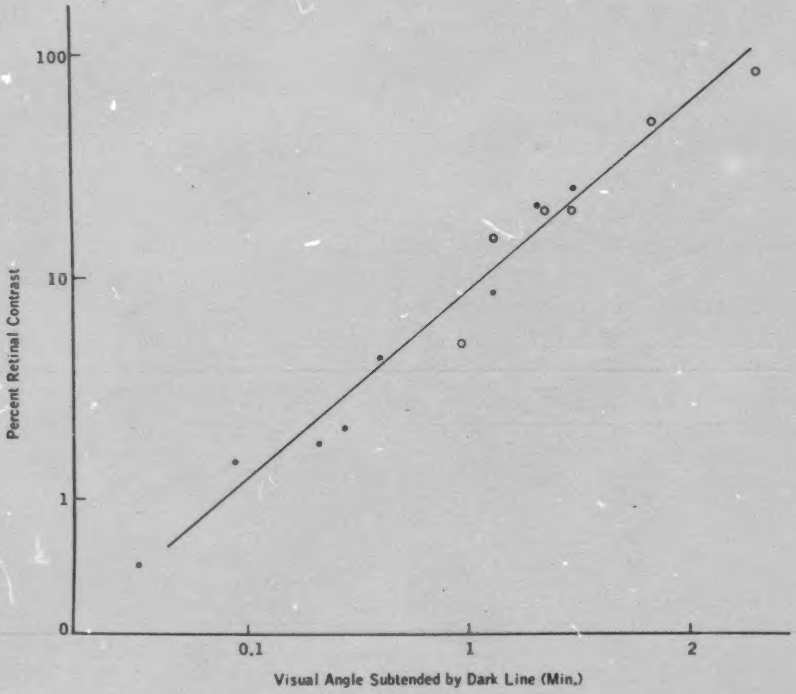

FIGURE 4 Log percent retinal contrast measured as a function of log visual angle (in minutes of arc) from data of DeMott[7] and Westheimer and Campbell.[21] Data are from the steer eye (open circles) and human eye (dots). Replotted from Boynton.[5]

reduction of visibility. The bizarre inks and backgrounds now used for artistic purposes in some popular magazines sometimes produce very low physical and retinal contrasts and can thus be very difficult to read.

Despite the low retinal contrasts that fine lines produce, our ability to resolve such lines is remarkable. Under optimal conditions, a good observer can detect a line that subtends only 1/2 sec of arc,[10] which corresponds to seeing a wire only 1/16 in. in diameter at a distance of 1/2 mile! Extrapolating from Figure 4, it can be estimated that that corresponds to a retinal contrast of less than 0.01%, and that is based on an illuminance distribution with gradual contours. It is probable that the contrasts plotted in Figure 4 are too low, because of difficulties of experimental measurement in both experiments and the double-traverse of the light through the eye in the human measurements. But even if the retinal contrasts were as much as 10 times as high as this figure shows, the retinal contrast at the threshold of detection is still very low.

A PRIORI *EXPECTATIONS CONCERNING RETINAL MECHANISMS*

From the foregoing, it should be clear that one of the most critical problems that the visual system meets, and somehow solves, is the detection of very low contrasts on the retina. If each retinal receptor had a private pathway to the brain, then very small differences in the initial signals produced in adjacent or nearby receptors would require preservation all the way to the brain for the difference to be discriminated there. Because noise is inevitably introduced in each stage of any information-transmission system, including a biologic one, a small difference would undoubtedly be obscured by noise by the time the original activity expressed itself in the brain. For this reason, we would expect *a priori* to find neural mechanisms to detect and augment the differences in signal strengths near the receptors. Furthermore, it would seem helpful to involve a large population of receptors to increase the statistical reliability of the difference. That the detectability of a line of fixed width has been found to be critically dependent on its length suggests that this is so.

CONTRAST DETECTION AND LIGHT INTENSITY

Although retinal contrast does not depend on light intensity, the threshold of detectable contrast does. We are all familiar with this from everyday experience. The physical contrast provided by, say, a newspaper depends only on the reflectances of the paper and ink used, and not on the level of illumination of the newspaper; nevertheless, we know perfectly well that it is difficult to read the fine print in dim light, and under still less favorable conditions—for example, under the light of the moon—only the largest headlines can be resolved.

The most extensive data related to this matter have been concerned with the detection of circular patches of light against a uniform background of variable luminance. When the test spot is very small, the task has much in common with what is involved in the resolution of fine detail. From extensive data collected by Blackwell and McCready,[4] I have selected the values shown in Figure 5. A test spot subtending only 1 min of visual angle and lasting for only 1 msec was used. The function labeled $\Delta L/L$ shows that the contrast required just to see the test spot drops from more than 1000% at the lowest luminance used to slightly more than 1% at the highest.

The same data can be plotted in another way. The curve labeled ΔL shows the just-visible (threshold) increment of luminance provided by the test spot. At very low levels, this threshold increment is independent of background luminance over a range of luminances that, although very dim, allow distinct visibility. As the log of background luminance (in footlamberts) is increased to more than −2, the threshold rises—slowly at first, accelerating gradually, and finally approaching a unit slope (corresponding to a $\Delta L/L$ slope of nearly zero).

Many other detectors of contrast, such as photographic plates and television pickup tubes, behave somewhat similarly. Like the eye, they

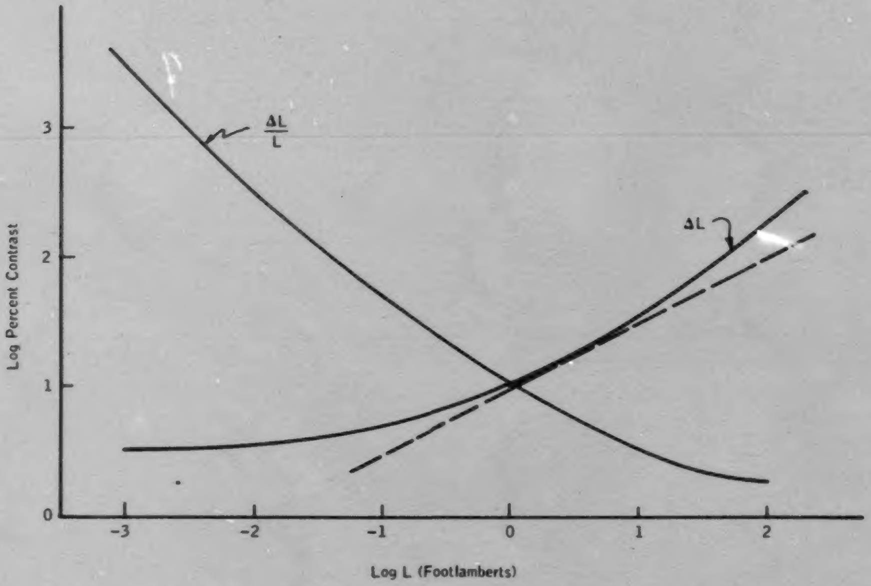

FIGURE 5 Contrast thresholds ($\Delta L/L$) as a function of log background luminance, for a 1-min spot exposed for 1 msec (data from Blackwell and McCready[4]). Also shown is the corresponding curve for the increment threshold, ΔL. For this curve, the values on the ordinate are in log fl. The dashed curve shows the predicted behavior of an ideal detector.

perform better when there is more light. Ideal detectors also behave this way: as the numbers of photons increase, so does the statistical evidence available to discriminate an increment from its background. The dotted line in Figure 5 shows what is predicted for the ΔL curve of the ideal detector. This line has a slope of exactly 0.5 and may be extended indefinitely in both directions.

This comparison shows that the eye is not an ideal detector. For the conditions shown in Figure 5, in which a very small and brief test spot was used, optimal performance is achieved at a log background luminance of about 0.5. At luminances both below and above that, more light is required in the increment spot than the ideal-detector model would predict. At low levels, this has most often been explained by postulating an intrinsic noise of the retina. It causes a minimal value of ΔL to be reached (about 7 fl in this case) at low background levels; the value would be the same in total darkness. In other words, this much luminance is required for the test spot to produce a signal in the visual system that can be discriminated from random background activity that exists in total darkness. At high background levels, the eye is also responding less efficiently than it might, probably because of the influence of various adaptive mechanisms whose function is to prevent saturation of the signals being transmitted through the visual system.*

RETINAL CONTRAST VERSUS SUBJECTIVE CONTRAST

One further psychophysical observation bears reporting before we turn to physiologic mechanisms: the highly nonlinear relationship between retinal contrast and its visual effects.

This relationship is most easily examined in a large bipartite field, where physical contrast and retinal contrast are essentially the same. The nonlinearity first expresses itself in the fact that, for very low objective contrasts, no border is seen. With further increases in contrast, the border will become visible, and subjective contrast will then increase very rapidly at first, and then more slowly. For an objective contrast of more than 20% or 30%, the border between the half-fields has already become so vivid and distinct that subjective contrast will not increase much further; further increases in physical contrast produce only very small additional increases in subjective contrast. We have attempted to measure this in the laboratory, using psychologic scaling methods. It is most difficult, because the observer cannot ignore brightness differences that are needed to produce, and therefore are correlated with, physical contrast. Figure 6 is a schematic representation of the relationship between objective and subjective contrast.

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*Further information on the effect of light intensity on contrast detection can be found in papers by Jones,[12,13] Barlow,[1] and Rushton,[20] which also contain additional references.

A more objective way to demonstrate this relationship is to make some measurements of visual performance as a function of contrast. Some years ago, we measured the ability of observers to seek out and recognize complex critical targets presented against a background of pseudo-targets. Figure 7 shows that the main improvement in visual performance is associated with the lower range of physical contrasts.

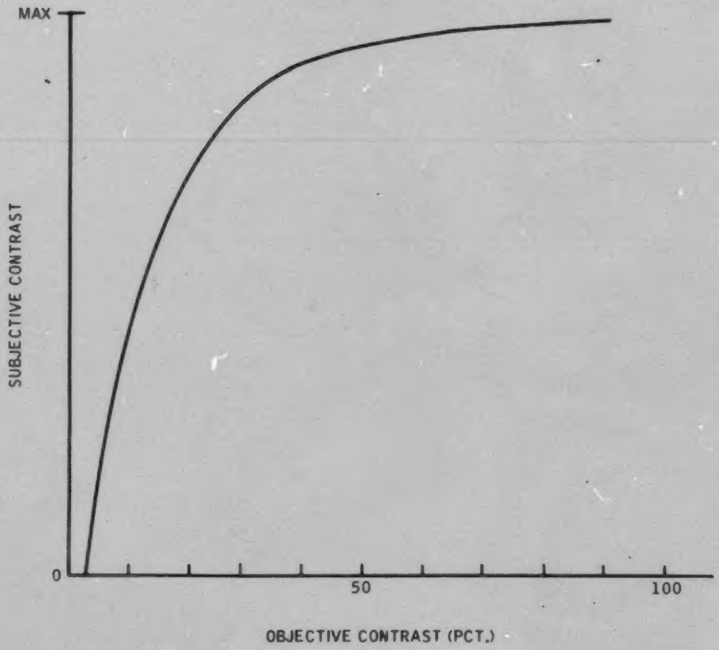

FIGURE 6 Schematic representation of the relationship between objective and subjective contrast

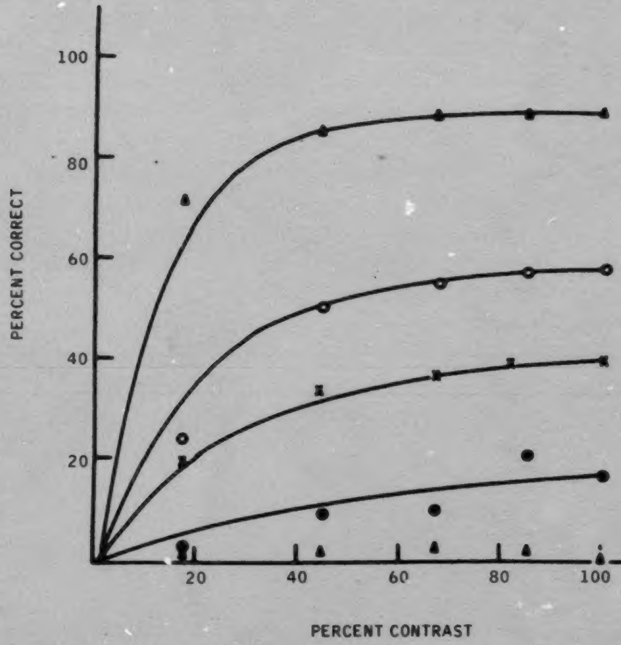

FIGURE 7 Percentage correct target identification by five subjects, in a complex search task, as a function of the contrast between the stimuli searched and the background against which they were presented. (Reprinted from Boynton and Bush.[6])

RETINAL ANATOMY

We will now review some of the evidence that clearly tells us that the outputs from the retinal receptors become highly interconnected as information is processed in the retina.

Consider first the anatomy of the retina. In the human eye, there are about 125 times as many receptors (rods and cones) as optic nerve fibers. Such convergence constitutes one basic form of retinal neural interaction, and it is a principal determinant of the summative receptive field. If many receptors feed information to the same optic nerve fiber, the results of feeble excitation in the individual receptors (each too weak to evoke sensation) can summate, vastly improving the probability that information will be delivered to the optic nerve fiber and thence to the brain. That is accomplished, however, at a sacrifice of visual resolving power.

Convergence is very nonuniform and depends greatly on the part of the retina stimulated. In the peripheral retina, many thousands of receptors deliver their outputs, via the intermediary bipolar cells, to a single ganglion cell. The central retina is very differently organized. When we fixate on a stimulus, we move our eyes to place its image in a highly specialized region of the retina, the fovea centralis. Only cone receptors are found in this region, and it is here that our visual acuity is by far the best. The convergence ratio in the fovea is about unity. This has sometimes been interpreted to mean that each foveal receptor has a private pathway to the brain. That is a mistaken conception; on the basis of the arguments presented above, we would not expect such private pathways, and the direct evidence now to be reviewed shows that we do not find them.

Anatomic evidence shows many interconnections among the retinal pathways, including those which begin in the fovea. On the basis of light microscopy, it has long been known that, in addition to convergence where it occurs, the human retina is richly supplied with cells that do not appear to be involved in the direct transfer of information from receptor to bipolar to ganglion cell (the classical visual pathway in the retina), but that seem to exist specifically to provide lateral interconnections between the neurons of the basic pathways. An excellent notion of this may be gleaned by examination of Figure 8, a schematic diagram of the retinal connections, based heavily on recent evidence provided by electron microscopy. Note that there are two classes of cells that provide lateral interconnections. Horizontal cells (H) interconnect the rod and cone receptors; they are believed to receive information from and feed it back into the receptors. Deeper in the retina, amacrine cells (A) interconnect bipolar and ganglion cells; they are also interconnected with one another and thus have the potential to carry information over very long lateral distances in the retina. The receptors themselves are also in intimate contact, with so-called tight junctions between rods and cones and between cones and cones.

The details of retinal anatomy vary from one species to another, but all eyes that have been studied so far have sufficient lateral connections to produce substantial lateral interaction effects. The list includes the lowly horseshoe crab, *Limulus*, long a favorite specimen for visual investigation. This animal has a faceted, compound eye; each ommatidium

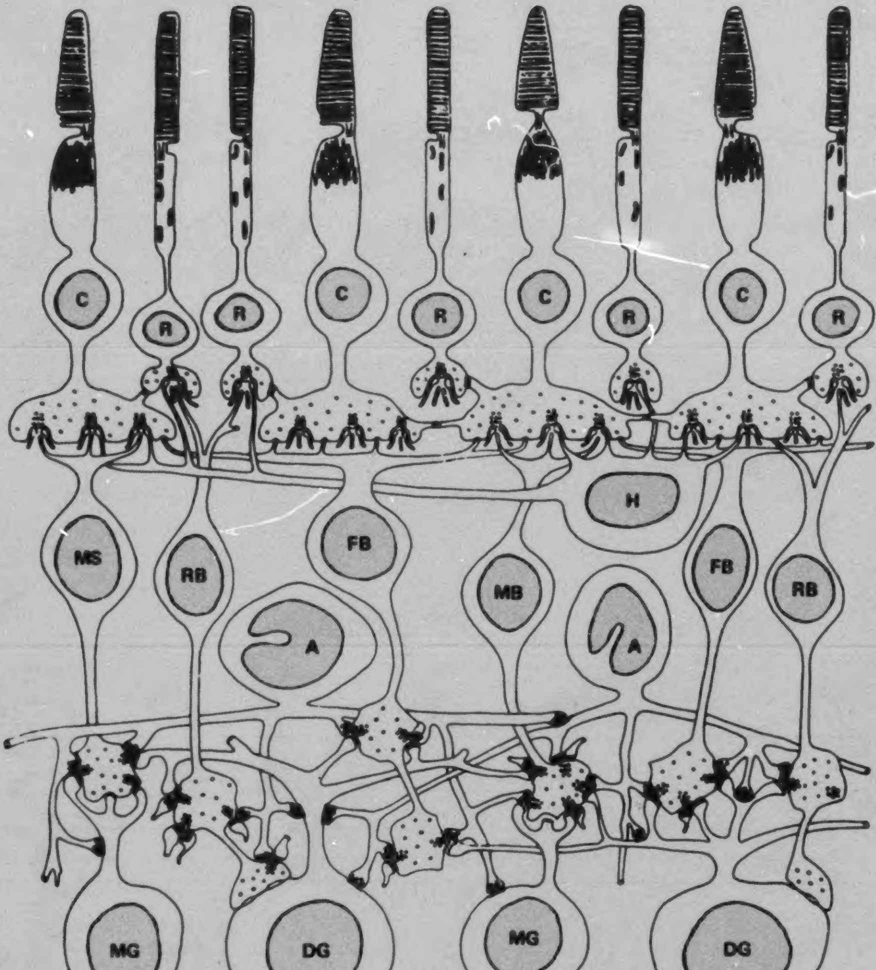
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FIGURE 8 Summary diagram of the interconnections among the neural cells in the retina. A, amacrine; H, horizontal; C, cone; R, rod; MB, midget bipolar; RB, rod bipolar; FB, flat bipolar; MG, midget ganglion; DG, diffuse ganglion. (Reprinted with permission from Dowling and Boycott.[8])

connects primarily to a single optic nerve fiber. Hartline first chose it, many years ago, because he believed it to be free of complicating lateral interconnections. But he and Ratliff[9] have since shown that a lateral plexus of fibers exist that interconnect the pathways, and furthermore that these connections have an exclusively inhibitory function. This work, described in detail in Ratliff's fine book, *Mach Bands*,[17] provides some of the clearest available illustrations demonstrating that these interconnections provide a neural mechanism for contrast enhancement.

INHIBITORY MECHANISM IN LIMULUS

An experimental arrangement of Hartline and Ratliff,[9] using the eye of *Limulus*, is shown in Figure 9. Light A can stimulate only ommatidium A. In the absence of other stimulation, this produces a particular frequency of firing in the optic nerve fiber A. Light B stimulates only ommatidium B and produces a particular rate of discharge in fiber B. Suppose that, with light A turned on at its original intensity, a light stimulus to B is provided also. At a very low intensity of stimulation of B, the result will be no firing of fiber B, and no effect on the firing rate of fiber A. But, as the intensity of stimulus B is gradually increased, fiber B begins to fire.

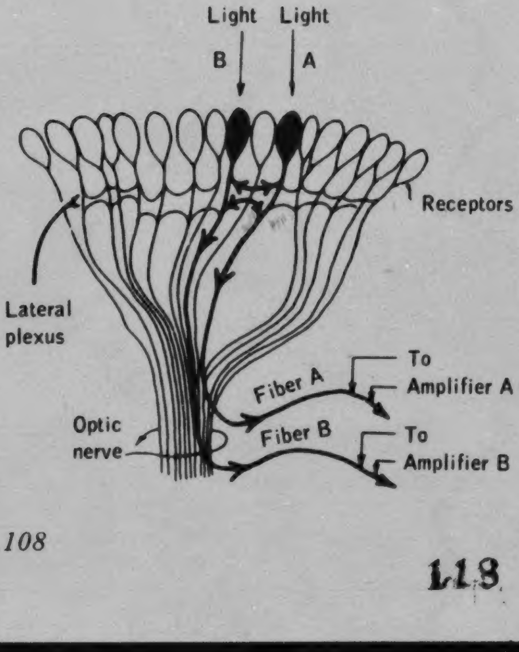

FIGURE 9 Experimental arrangement of an experiment by Hartline and Ratliff (1957), designed to show lateral inhibition in the eye of *Limulus*. (Adapted from Ratliff.[17])

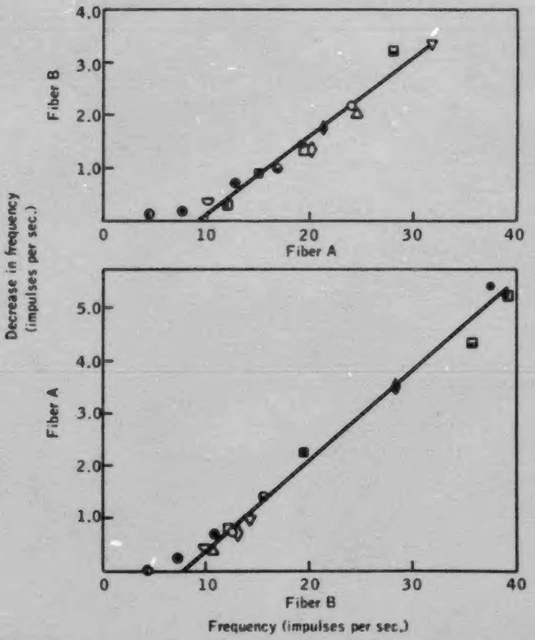

FIGURE 10 Top, inhibition in fiber B produced by activity in fiber A. Bottom, converse inhibitory action. (Reprinted with permission from Hartline and Ratliff.[9])

As shown in the lower graph of Figure 10, this activity in fiber B is associated with a decrease in the frequency of firing in fiber A, which is still being directly activated only by the original light intensity delivered to ommatidium A. It turns out that, once some threshold frequency is reached, the inhibitory effect of B (as measured by the decrease in frequency of firing in fiber A) is linearly related to the frequency of firing of fiber B. It is therefore concluded that the B system is doing the inhibiting, with the strength of the inhibition depending on the rate of activity in the B fiber. The experiment can also be done the other way around, and it is found that activity in fiber A produces a reciprocal inhibitory effect on the normal firing of fiber B (upper graph of Figure 10).

The nature of the inhibitory effect depends also on the distance between the ommatidia being stimulated. In Figure 11, inhibitory effects are measured in turn from fibers B and C, in response to variable intensities of illumination of ommatidium A, the latter leading to a variable frequency of response in fiber A, as shown on the abscissa. During recording from fiber B, no inhibition occurs until there are about five im-

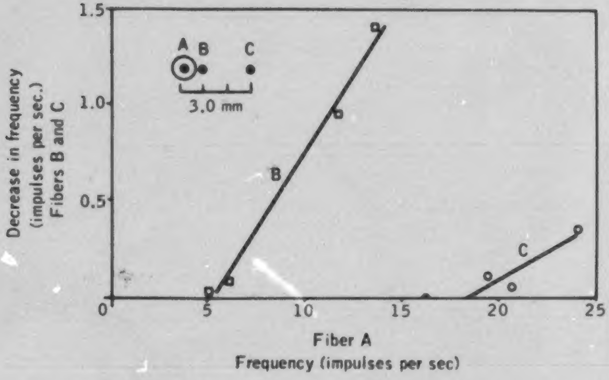

FIGURE 11 Experiment to show inhibition of activity in fiber A, produced by nearby stimulation at B and more remote stimulation at C. (Reprinted with permission from Ratliff.[17])

pulses per second in fiber A; inhibition then rises rather steeply with increasing frequencies of A. During recording from fiber C, which is more remote from A than is B, the threshold is reached at much higher firing levels in A (nearly 20 impulses per second), and inhibition increases more gradually than for fiber B.

It is to be emphasized that the inhibitory action associated with a particular system, such as the A system of the previous example, depends only on the frequency of response of that system, without regard to how the frequency was produced. An interesting example of this is an experiment on disinhibition shown in Figure 12. Here, three stimulus fields—A, B, and C—are used, with recordings taken from fibers A and B. Stimulation of A alone produces an intermediate discharge rate, shown on the left side of the bottom record. Field C, at the intensity used, is too far from A to have any effect, as shown in the middle part of the lower record: when C is turned on, the frequency of discharge in A continues much as before. The left side of the upper record shows what happens when ommatidium B is stimulated along with ommatidium A. The stimulus to B is strong enough to produce a high frequency of discharge in ommatidium B, which in turn is associated with a marked inhibition of the firing rate in A. If field C is turned on, it produces a marked inhibition of fiber B, as reflected in the middle part of the uppermost record. This reduction in the response rate of fiber B releases in turn some of the original inhibitory effect of B on A, as revealed by the fact that, while field C is turned on, the response to A increases.

Note that the intensity of stimulation of B has not been varied in this example. The response to a fixed intensity of stimulation of B has been modified by the action of C. The same effect could have been produced simply by reducing the intensity of stimulation of B. This example indicates that it is the *response* to B that is the simplest variable to consider in predicting the inhibitory action of B on A, rather than the *intensity* of the stimulus to B.

As stated previously, these inhibitory interactions are reciprocal. If A inhibits B, then B also inhibits A. But the inhibitory action of B on A is less, because of its inhibition by A, than it would be otherwise—and so on. Consequently, inhibitory relationships that are relatively easy to understand in terms of response rate become difficult to calculate on the basis of stimulus intensity patterns alone. The equations required to do this have been worked out by Ratliff, and their predictions have been tested in direct experiments. In one such experiment, Hartline and Ratliff[9] were able to show clearly that neural gradients are sharpened and enhanced by the inhibitory process. Moreover, the subjective phenomena of Mach bands, found in human observers, can be accounted for quantitatively in terms of the inhibitory relationships worked out in *Limulus* (see Figure 13). This is very important, because it strongly sug-

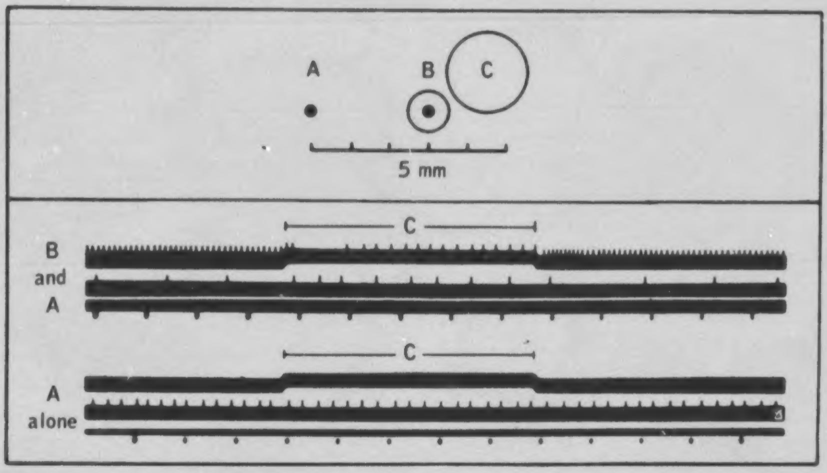

FIGURE 12 Oscillographic records of electrical activity of two optic nerve fibers (A and B), showing disinhibition. (Reprinted with permission from Ratliff.[17])

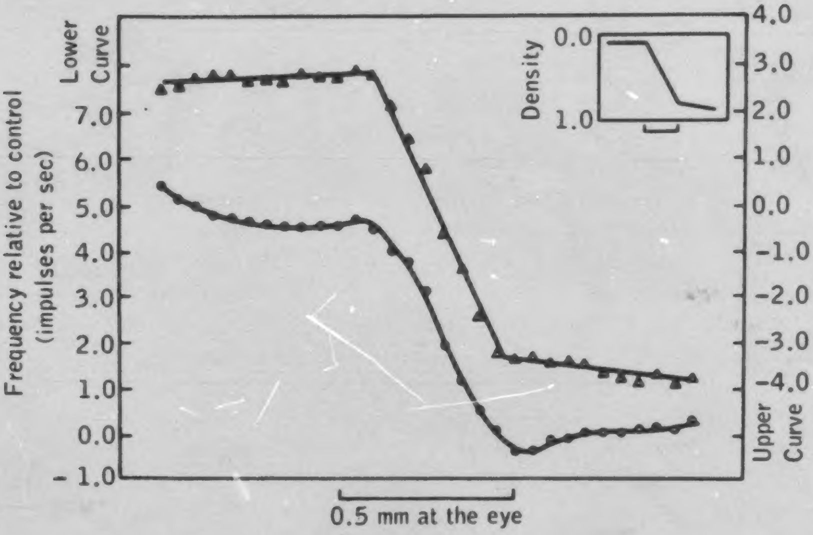

FIGURE 13 The upper record was obtained when the stimulating light was allowed to illuminate only one ommatidium from whose optic nerve fiber the records were taken. The stimulus was a lamp, having a light distribution as shown in the inset, moved laterally across the ommatidium (X-axis). When all the ommatidia were uncovered and the stimulus was moved laterally as before, the lower record was obtained. Note the evidence of overshoot and undershoot near the top and bottom of the curve. This corresponds roughly to the appearance of Mach bands by human observers when they view such a stimulus. (Reprinted with permission from Ratliff and Hartline.[18])

gests that the kinds of inhibitory effects found in *Limulus* also occur in the human retina.

RECEPTIVE FIELDS AND TRIGGER FEATURES

In addition to the purely summative types of receptive fields already mentioned, which are found in the dark-adapted eye, much more complex arrangements are typical in warm-blooded vertebrates. Figure 14 shows one example. A microelectrode is plunged into the cat retina through the front of the eye and is in contact with a single retinal ganglion cell whose axon forms an optic nerve fiber. The experimental arrangement allows light spots to be flashed on and off in various parts of the visual field. A central area is found where the onset of a light spot produces an increased discharge in the ganglion cell (an ON response), whereas the turning off of the light yields no response. In the horizontally shaded area shown surrounding it, both ON and OFF responses are obtained. In the outer field (diagonally shaded), only OFF discharges can be recorded.

This arrangement of an excitatory center field and inhibitory surrounding field has been found in many other experiments. (The excitation–inhibition relationship is often reversed.) In the warm-blooded vertebrate, unlike *Limulus,* it is found that the retinal ganglion cells respond at a modest rate, even in the absence of light stimulation. Thus, inhibition can also reveal itself as a reduction in the resting rate of response during a period of prolonged stimulation.

In the frog, Lettvin *et al.*[16] were the first to show that a single ganglion cell responds to remarkably specific trigger features of the stimulus, depending on the cell recorded from. In some cases, for example, a unit will respond well only to convex dark spots moving in very particular ways, and to no other stimulus. As Lettvin *et al.* remark, it is tempting to call such a specialized unit a "bug detector." More recently, the name "trigger feature" has become associated with the peculiar, com-

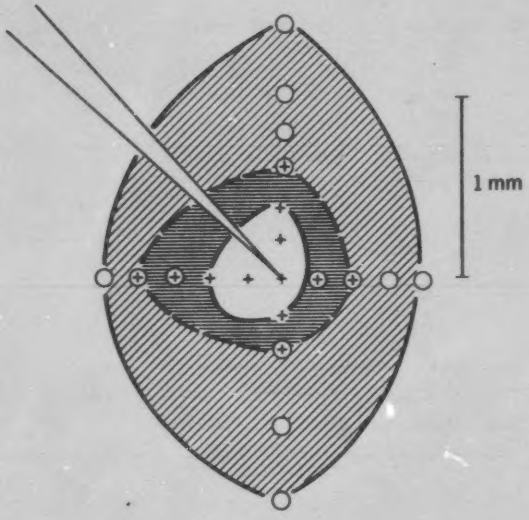

FIGURE 14 Receptive field of a cat ganglion cell, obtained by Kuffler.[14] This unit has an ON center and an OFF surround. (Reprinted with permission from Riggs.[19])

plex aspects of a visual stimulus that are often found necessary to trigger activity in a single unit in the retina or higher visual center.

Trigger features have been examined in detail in the primate visual cortex, especially by Hubel and Wiesel in a long series of experiments (see Hubel[11]). Although consideration of the cortex is outside the boundaries of this presentation, it is significant to note that the center–surround relationships that are found when recording from primate cortical cells are nearly rectangular, rather than circularly organized. These cells are often most sensitive to moving lines, and typically are directionally sensitive. In the optic nerve fibers of spider monkeys, only the concentric ON–OFF, center–surround arrangement was found at this level of the primate visual system.

Directionally sensitive units have been found in the retina of the rabbit by Barlow and Levick.[2] Some of the results from one of their experiments are shown in Figure 15. A black edge is caused to move across an illuminated slit. When the slit width subtends 34 min, the directional effect is very clear. When the slit is closing in a preferred

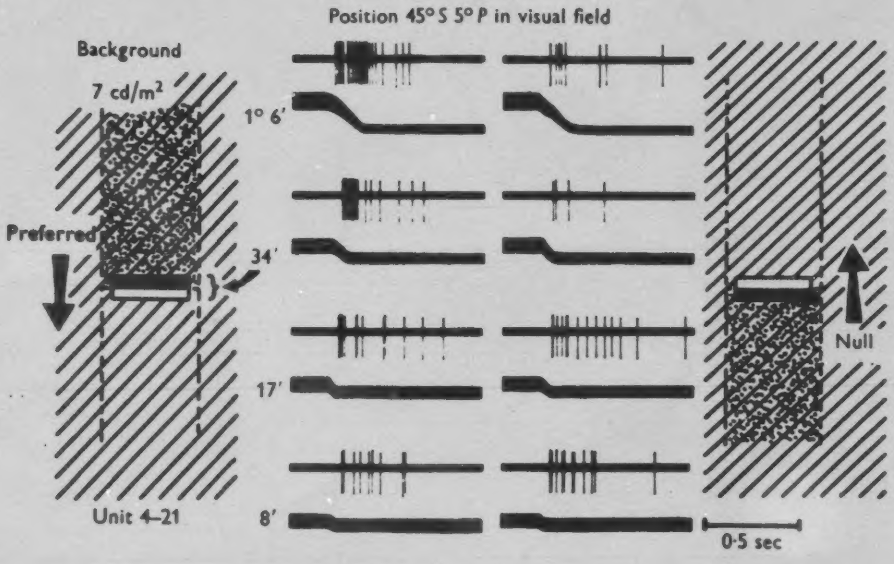

FIGURE 15 Responses to the motion of black edges across slits of various widths. (Reprinted with permission from Barlow and Levick.[2])

direction (from top to bottom, as shown on the left side of the figure), the retinal ganglion cell under investigation responds with a vigorous ON-burst. But when the experiment is repeated exactly, except that the slit closes from bottom to top, only a very weak response is recorded. When the slit width is only 8 min, no significant directional effect is obtained. This suggests that the directionally sensitive mechanism must be contained in a larger retinal region. These investigators have done a good deal of speculation about the retinal interconnections responsible for such behavior; the horizontal cells of the retina have an important role to play in their schema. It should be emphasized that the temporal characteristics of retinal responses are critical in such a system. These have been investigated in some detail in further work of Barlow and Levick, as well as in *Limulus* by Ratliff.

The electrophysiologic work carried out to date has consistently involved the use of very-high-contrast visual stimulation. It is not easy to relate the psychophysical results described in the first part of this presentation to the electrophysiologic data discussed in the last. The work of Hartline and Ratliff is clearly the most relevant in a formal sense, in that it deals explicitly with contours and shows how the visual nervous system can operate to enhance them. Although it is a long way from *Limulus* to man, there is strongly suggestive evidence that similar mechanisms operate in the human eye.

It has perhaps not been emphasized enough that these inhibitory mechanisms appear, on the basis of both behavioral and electrophysiologic evidence in vertebrates, to be specific to the light-adapted eye. In the dark-adapted state, in which the task of the eye is to gather as many photons as possible, receptive fields are purely excitatory. When more light is available, the inhibitory mechanisms come into play, and they are clearly implicated as mechanisms of contrast enhancement that help in the perception of fine detail.

I wish to thank Thomas R. Corwin for his critical reading of this manuscript.

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DISCUSSION

DR. BOYNTON: I hope that this story has left the correct impression that the business of dissecting the retinal image, generating the neurologic code, and then transmitting this information to the brain is not a set of passive mechanisms. The code is related to what has adaptive significance to the organism, and at many points along the way a very subtle difficulty with the pathways involved could conceivably result in improper functioning of these mechanisms.

DR. LINDSLEY: I am glad that Dr. Boynton brought up the spatial relationship of interaction within the retina. There is another important complexity: the temporal relationship of the stimuli. D. N. Robinson (Science 156:1263–1264, 1967) recently noted that the response of the eye to a second flash was masked, presumably through lateral inhibition, by what was contained in the first flash. He noted that a third flash, like your spatial elements in a section, would mask the second one in the same way that the second was masking the first. Thus, on the temporal side, there are phenomena somewhat similar to those on the spatial side. This adds another element of complexity.

Where reading difficulties are concerned, it might be better to focus on temporal than on spatial contrast. You can, in fact, combine them; that is, you can give stimuli both in sequence and separated in space. On the basis of what we know about lateral inhibition of the retina, there is a reason for what occurs between 60 and 90 days of age in an infant, as Robert Fantz shows (see p. 351) using a series of concentric circles or checkerboards. Why should concentric circles have a specific effect?

DR. BOYNTON: As far as I know, there is no explanation.

DR. SCHUBERT: In connection with perception of printed symbols, contrast would depend on illumination, and a number of figures are given by authorities as to how much light you need on the printed page. There is no consensus in this regard. How many footcandles are needed? Can any of the participants offer data concerning the contrast that is desirable in enhancing legibility?

DR. BOYNTON: There is a problem of definition that might be worth clearing up. When I used the term "contrast," I referred specifically to the physical, objective definition, the difference in luminance between two areas divided by the luminance of the larger of these two areas. Physical contrast so defined is independent of the illumination on a reflecting target, such as a letter on a page. The subjective "contrast" associated with this, however, is critically dependent on illumination level, and it increases with increasing illumination. There are probably dozens of mechanisms involved in this distinction.

As to what constitutes a proper level of illumination, I feel that, in spite of all the arguments and all the research, we do not know exactly what does constitute a proper illumination level. Obviously, it depends on the inherent contrast and size of the material being viewed, what is being looked at, and why. Blackwell (J. Opt. Soc. Amer. 36:624–643, 1946), on the basis of an extensive series of investigations at Ohio State University, has come up with a set of figures that have generated a good deal of controversy.

We keep learning new things. I was very impressed with what Dr. Young said concerning the effect of low levels of illumination on the development of myopia, which is ascribed to a great deal of activity in the accommodative mechanisms. If the illumination level is high, the depth of field of the eye is increased because the pupil is reduced. For this and probably other reasons, less accommodation is required at high illumination levels. Thus, there is now some evidence that tends to support the admonition, "Don't read in dim light or you'll ruin your eyes"–something my mother used to say to me. Possibly, she was correct in her assumption. But this is the first evidence that I know of to support this old wives' tale.

DR. BUSER: Concerning the degree of retinal inhibition: to effect that, the retina is active as well as passive. I think that there may be some very strong evidence of this. Take, for example, the directional-sensitivity cells, which are activated preferentially by a moving light. According to Barlow and Levick, special structures are very numerous in the retina of the rabbit, but the cat has very few; I do not know about primates.

DR. BOYNTON: I strongly concur that this is true. I tried to point out that there are species differences. I picked *Limulus* for a detailed illustration largely in deference to historical values: it was the first experimental animal in which clearly defined retinal interaction mechanisms were demonstrated, although many workers had felt for many years that they must be present in the human retina. Only within the last 15 years or so have they been found physiologically in any animal, let alone man. Your point is very well taken.

MATHEW ALPERN

The Pupillary Light Reflex and Binocular Interaction

I am going to discuss some recent experiments on one aspect of the cross-talk between the two eyes in what I am constantly reminded is thought to be the simplest of all reflexes, the pupillary reflex to light. I want to speak about binocular cross-talk in this "simple" reflex, as well as about some findings on a disorder of perception—something not associated with the reading problem in any proper sense of the term, but an interesting perceptual disorder nonetheless.

If all other conditions are equal, the pupil is always smaller when both retinas are illuminated than when one or the other is in the dark. Figure 1 shows some measurements of the size of the pupil in a junior medical student when one eye was in the dark and when both eyes were illuminated equally. Note that in the binocular case the pupil is always a bit smaller. By making a simple downward displacement of the monocular curves, one can get a fair prediction of the binocular data.

This is not the place to document the fact that the obvious sorts of artifacts that might account for this result—fusional movements, accommodative movements, and so on—do not play any role in the results of such experiments. Nor do I need to describe the experiments that show that it does not matter which retina is illuminated; both pupils always go together.

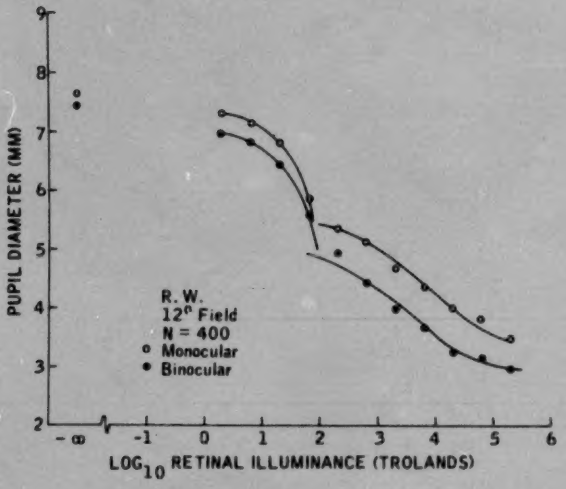

FIGURE 1 Diameter of the pupil of the left eye when both retinas are equally illuminated (dots) and when the left eye is in the dark (circles). The abscissa scale in each instance is the retinal illuminance (log trolands) of the right eye. (Reprinted with permission from ten Doesschate and Alpern.[2])

Figure 2 shows the nerve pathways involved. There are two places in the central nervous system that separately or together would allow added activity from each eye to pool in such a way that the binocular process produces a smaller pupil than the monocular. The places are at the level of the midbrain, and presumably nothing higher than the midbrain is involved.

The experiments illustrated in Figure 3 allow us to exclude two very simple ways in which the pooling might occur. We might assume that the nervous system responds to the light that goes to the left eye and adds it, as though the same light had been given to the right eye. In this figure, the dashed line is the predicted binocular pupil size according to this hypothesis. Clearly, it does not agree at all with the measured values (circles). Furthermore, the nervous system does not add the amount of contractions of the pupillary muscle. The solid line in the figure shows the expected result according to that idea. Simple addition, either of lights or of contractions, is an inadequate description of the results.

Although one can write the equations for this effect, the physiology cannot yet be said to be well understood. In the process of striving to build a reasonable model, Prof. J. ten Doesschate of Utrecht and I[2] stumbled onto something that might be of interest. We borrowed from a paper by Cooper *et al.*[1] Suppose you had an excitation pool (Figure 4) in the midbrain, and that the output of all the cells was determining the size of the pupil. Suppose that everything in the right circle were driven by the right retina and everything in the left circle by the left retina. (In binocular vision, of course, both systems respond.) This sort of scheme in a rough, qualitative way will account for the results we obtained.

This is purely speculative. Can we find any experimental evidence for this view? Perhaps we have a clue in an experimental finding of Hubel and Wiesel[3] on single nerve cells in visual cortex of kittens. Cutting the eye muscles of a newborn kitten results in an alternating divergent strabismus. After this strabismus had developed, Hubel and Wiesel measured the percentage of cells in the visual cortex that were binocularly driven. In the normal kitten—one with only a sham surgical procedure—

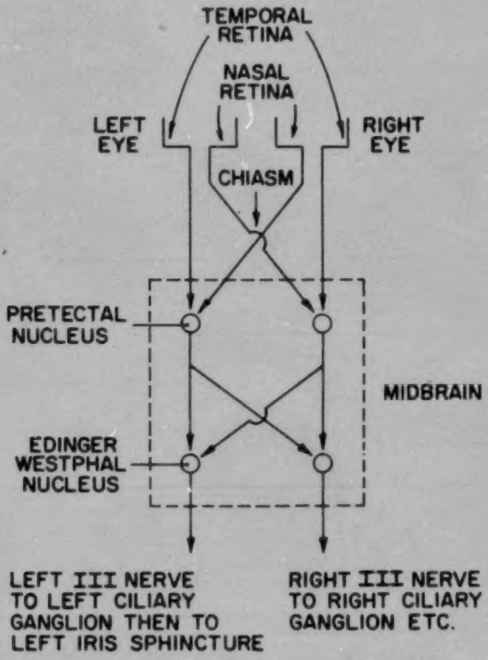
TEMPORAL RETINA
NASAL RETINA
LEFT EYE
RIGHT EYE
CHIASM
PRETECTAL NUCLEUS
MIDBRAIN
EDINGER WESTPHAL NUCLEUS
LEFT III NERVE TO LEFT CILIARY GANGLION THEN TO LEFT IRIS SPHINCTURE
RIGHT III NERVE TO RIGHT CILIARY GANGLION ETC.

FIGURE 2 **Black-box sketch of the nerve pathways for the pupillary light reflex.**

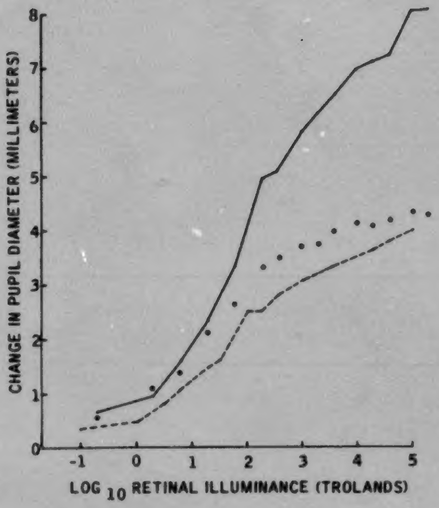

FIGURE 3 Change in diameter of the pupil of the left eye for equal binocular retinal illuminance. The circles represent empirical observations; lines are theoretical predictions based on the monocular measurements. The solid line is the change in size predicted if each monocular response is added linearly; the dashed line is the change predicted if the lights are added linearly. Neither fit is satisfactory. (Reprinted with permission from ten Doesschate and Alpern.[2])

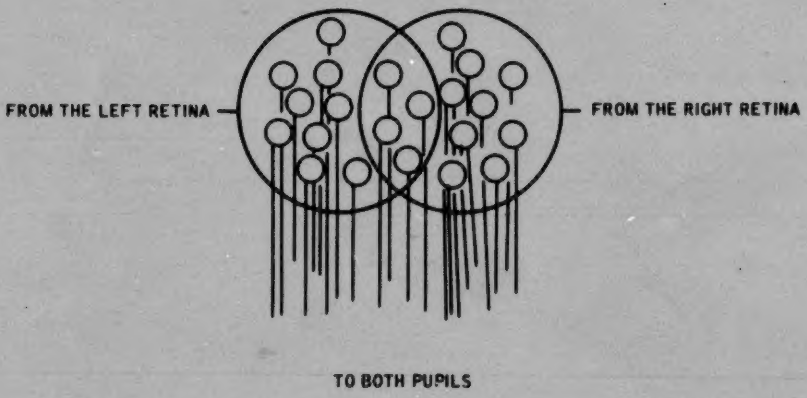

FIGURE 4 Scheme of midbrain pooling that could account for the fact that the pupil is always smaller when the retinas are equally illuminated, compared with the case when one retina is in the dark. The output of the pool drives both pupils, equally larger contractions being produced by having more cells responding. The cells contained in the right circle are excited by the right retina; those in the left circle are excited by the left retina.

a vast majority of the cells are binocularly driven. However, in the test kittens with strabismus, the matter is quite different. The vast majority of cells in their visual cortex are only monocularly driven. This looks like a paradigm for testing binocular pupil additivity. A person with alternating strabismus might be expected to show a much greater binocular additivity than in the normal eye (because fewer cells would be binocularly driven). As it happens, I have an alternating strabismus, so I put myself into this apparatus and did the experiment. It turned out to be nonsense: we could not get the expected results at all. The result was interesting, however. I would like you to think of the problem (a perceptual problem) of someone with strabismus whose eyes aim simultaneously at different parts of the visual world. Such a person's way of viewing the visual world binocularly is remarkably different from the way of a person with normal binocular vision. To avoid double vision with an alternating strabismus, the retinal activity of one of the two eyes must be turned down (if not off). In the clinical literature, this phenomenon is referred to as "suppression"; there is little concrete understanding of its physiology.

Figure 5 shows some of the measurements of the pupil (of my eye) in alternating strabismus. I was fixating with my left eye, although my right eye is the dominant one. Instead of getting the predicted superadditivity, there was no additivity at all. In binocular viewing, the pupils were the same size as when the right eye was in the dark. The nonfixating eye made no contribution to the binocular response. That was the first interesting aspect of our results.

The second was that, when the fixating eye was in the dark, the pupils were much wider than when the nonfixating eye was in the dark. In the normal subject, the eyes are equally effective in making the pupil smaller, irrespective of which one is fixating. But in strabismus, when the fixating eye (the left in Figure 5) is stimulated, the pupil is much smaller than when the nonfixating eye is stimulated. That is not a peculiarity of the left eye, but characteristic of whichever eye happens to be fixating at the time.

Figure 6 substantiates the last statement. When the right retina is fixating, it produces the smaller pupil. When the left retina is fixating, it produces the smaller pupil. It depends, not on which is the so-called better eye, as far as visual acuity or dominance is concerned, but on which of the two eyes happens to be fixating at the moment.

Having found that, I wanted to see whether there was a paradigm for

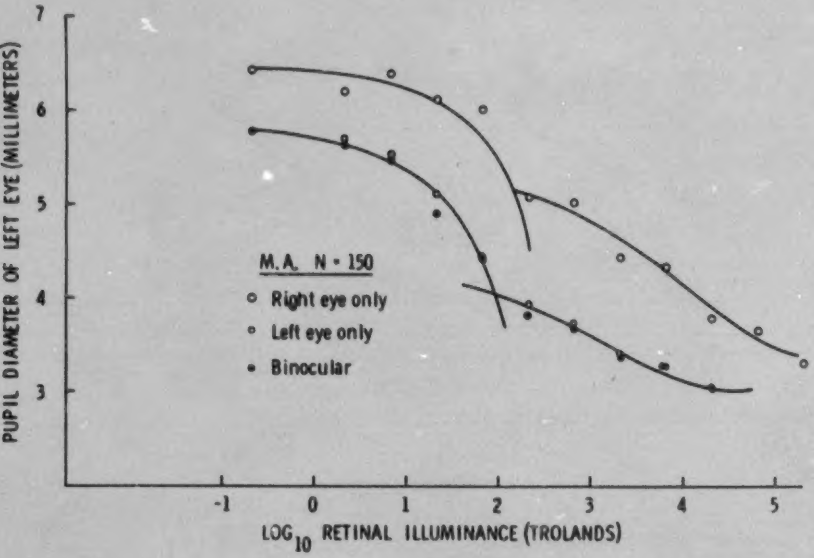

FIGURE 5 Size of the pupil of the left eye in observer M. A. (with alternating strabismus) as a function of the intensity of retinal illuminance. Left eye is always fixating. The dots show the results when the eyes were equally illuminated; the small circles, the results when the fixating eye was illuminated; and the large circles, the results when the squinting eye was illuminated. (Reprinted with permission from ten Doesschate and Alpern.[2])

a similar effect in normal eyes by making measurements that involve the classical experiment of retinal rivalry. Briefly, one puts overlapping contours onto the two retinas (for example, vertical black and white stripes seen by the left, horizontal stripes by the right). The result is that these conflicting borders cannot be unified into a perfectly consistent whole. Normal observers alternately hold clear first, say, the vertical lines, and then the horizontal lines, oscillating from one to the other. I thought that perhaps the phenomenon of turning down or turning off the retinal input to the pupil, which occurs in strabismus, would also show up on normal eyes, if they were forced into this rivalry situation. In fact, it looks as though it does.

The experiment is tricky, because one must remember that in the normal eye each retina is driving both pupils to the same extent. If the subject is presented with the overlapping contours, shown in Figure 7, and the illumination on the two eyes is the same, it does not matter

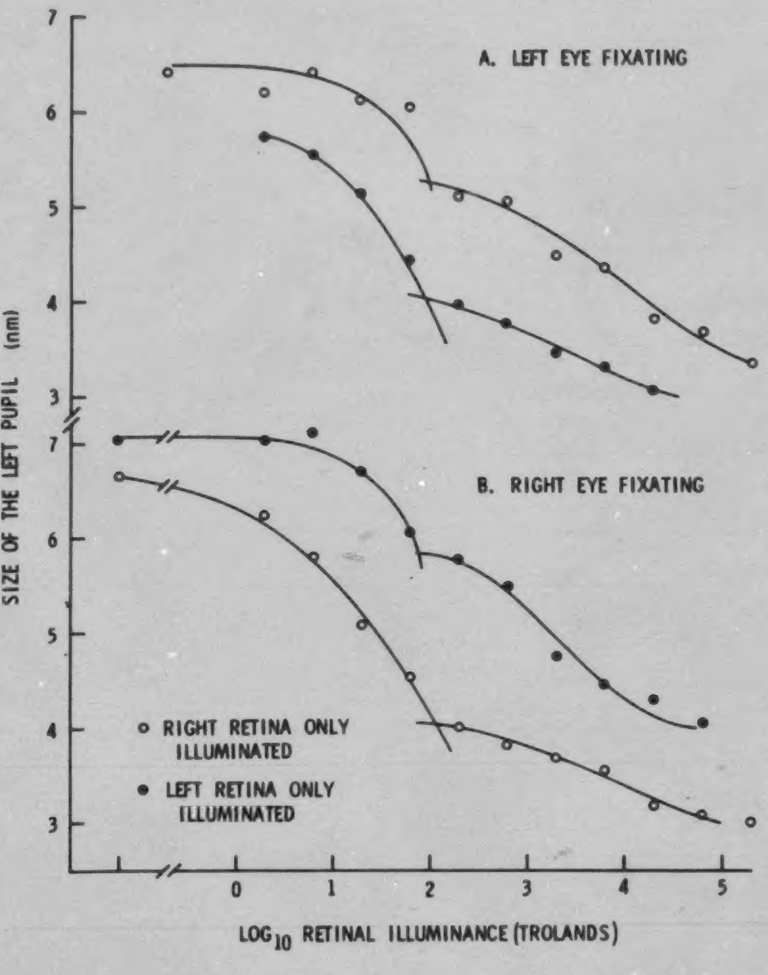

FIGURE 6 The amount of pupil contraction evoked in alternating strabismus depends on which retina is fixating. Illuminating the fixating eye always produces the smaller pupil.

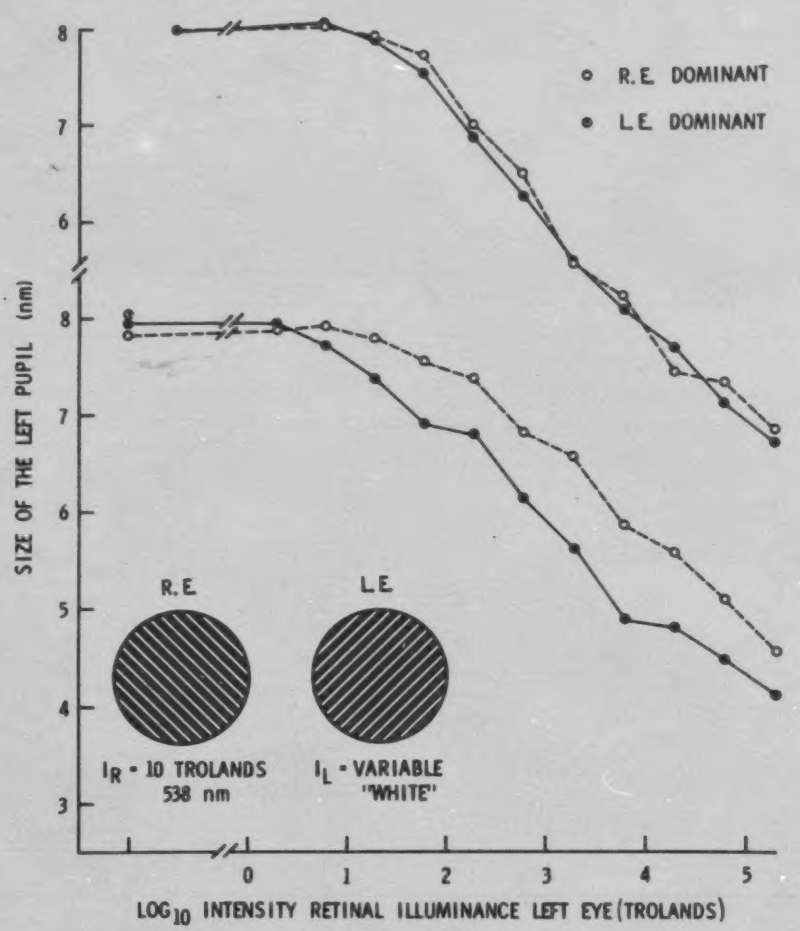

FIGURE 7 The pupil size (of the left eye) evoked by different amounts of retinal illumination in the normal eye during a retinal-rivalry experiment. The abscissa shows the intensity of retinal illuminance of the left retina. The circles represent measurements made while the right eye dominated, the dots, those made while the left eye dominated. Upper, results when the two retinas were always illuminated the same amount. No differences in the responses were measured when the data were obtained in phase and out of phase with the illumination of the dominant retina. Lower, results when the illumination to the right retina was held fixed (green light of 10 trolands) while that to the left retina was varied. The pupil is smaller when the more intensely illuminated retina is dominant than when the more weakly illuminated retina is dominant.

which retina dominates; either will give the same result. Thus, the top graph in Figure 7 shows the same pupil size, regardless of whether the pupil is photographed in or out of phase with the rivalry. No difference would be expected, because the illumination on each retina was always the same. To bring out the influence of rivalry, one must introduce a difference in the retinal illumination in the two eyes.

The experiment is straightforward, and the results are illustrated in the bottom graph of Figure 7. We hold the retinal illuminance of the right eye fixed and vary the intensity of illuminance of the left retina only. The right eye views green light of 10 trolands that is not very different from the intensity of illuminance of the low levels used to stimulate the left retina, and the result—as we have already seen—is that there is no difference in the size of the pupil in the left eye, whether the left or the right retina dominates. However, as we increase the intensity of illuminance to the left retina, so that it is appreciably greater than that of the right, it matters a good deal which retina is dominant. At any abscissa setting, the intensities of illuminance to the two retinas do not change; but the pupil is much wider when the retina receiving the weaker light is dominant than when it is not.

In general, the phenomenon that we are trying to understand in strabismus seems to have a counterpart in the normal eyes' viewing a rivalry target.

I do not fully understand the implications of what I have described. This is, in many ways, a very surprising result: where vision is suppressed by the dominance of one retina, photopupillary motion is also suppressed.

One suggestion is that whatever is turning down this visual impression is turning it down at the cortex. If so, it must also send separate signals down to the midbrain level to turn down the photopupillary response. Alternatively, perhaps the turning down is going on at the retina itself, in which case a separate turning down for the pupil is not necessary, because vision and photopupillary motion are probably mediated by the same nerve pathways at the level of the retina. Either suggestion is rather disturbing, and the evidence for each is not very impressive.

What is the relationship of these ideas to the problems of reading? In the process of reading, one makes eye movements of very high velocity, saccades; in making a saccade, the visual system also undergoes a momentary turning down of activity (it takes more light to produce a threshold than when the eye is immobile). It has been speculated that the turning down of the sensitivity of vision during saccadic eye movements resembles the turning down of vision in retinal rivalry, and the same sort of turning down that is found in strabismus. I find it difficult to conceive a good experiment that might accumulate evidence to support such ideas.

I am indebted to Dr. Keith Burnes and Joel Sugar for their able technical assistance in some of the experiments discussed here.

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DISCUSSION

DR. WADE MARSHALL: Curt Richter (Johns Hopkins Med. J. 122:218–223, 1968) recently reported an interesting study of alternating strabismus on 24-hr cycles. This is such a curious phenomenon that I wrote to him, and he replied that he had checked 30 cases in which it operated like clockwork, the disturbance tending to improve as the patient got older. Psychiatrists tell me that they sometimes see 24-hr cycles in manic-depressive patients.

DR. ALPERN: The phenomenon of periodic strabismus, in which the patient is normal some of the time and has strabismus some of the time, is very well known. I did not know that the alternation can occur with such beautiful regularity.

The mechanism of turning down vision during a saccade is not a simple matter. There is a large literature concerning whether saccadic suppression occurs at all. Evidence is accumulating that the output from the retina is not completely turned off during the saccade. However, the activity is clearly reduced (because of the extremely high velocity of eye movements), and there is also some reduction in visibility. The best data I know of are those of Riggs and his students, Amy Schick and Francis Volkmann (J. Opt. Soc. Amer. 58:562–569, 1968). There does seem to be a genuine reduction of sensitivity that is not due to any of a number of sources of artifact. Zuber *et al.* (Exp. Neurol. 14:351–370, 1966) have found evidence of the suppression of pupillary light response during the saccade, but there are still some problems in understanding the phenomenon.

DR. INGRAM: Is there any evidence concerning persons with constant nystagmus while reading? How do they manage to read, to get a retinal outline down?

DR. ALPERN: I have no evidence on this except the subjective reports of a professor of mathematics at the University of Michigan who has very poor visual acuity and a constant nystagmus. He reported that, when he was observing the rotating turntable of his phonograph, which had a speed of rotation in phase with his nystagmus eye movements, the phonograph record on the turntable appeared stationary. That is the only evidence I know of.

DR. SILVER: Did you notice a difference in position or shape of the pupil? Sometimes on a flash of light, the pupil becomes eccentric, and I wondered whether you had seen that.

DR. ALPERN: These studies were not done with flashing lights, but with an optical system presenting a binocular Maxwellian view. The pupil was photographed in the steady state under infrared light with infrared film. There may be such effects, but I would not have seen them in these experiments.

DR. LINDSLEY: You referred to retinal suppression in Riggs's experiment.

DR. ALPERN: I did not mean to. There is a saccadic suppression, which Riggs does not for a minute believe is in the retina. If I were to infer what Riggs thinks, saccadic suppression is in the cortex, and I have no way of knowing whether suppression is in the cortex in this other system responsible for the observations I have presented. It is a bit awkward, because multiple connections are needed, not only between the cortex and retina for vision, but through the pretectum for photopupillary motion. It is not obvious why anyone would build a railroad that way. However, if saccadic suppression were retinal or if this suppression in rivalry were retinal, then it would not be at all surprising for both vision and photopupillary response to be suppressed. That would be the logical consequence of the neuroanatomy, inasmuch as in every way that we can make the tests it is evident that, within the retina, the nerve pathways for vision and photopupillary motion are identical.

DR. LINDSLEY: There is already some evidence. Dr. Buser and his co-workers (C. R. Soc. Biol. Paris 154:38–42, 1960; J. Neurophysiol. 26:677–691, 1963) have shown that there are pathways to and from not only the tectum, but also the lenticular formation.

DR. ALPERN: But why would anyone want to develop a special pathway from cortex to midbrain just to turn down the pupillary response to a binocular rivalry stimulus?

MITCHELL GLICKSTEIN

Neural Organization in Vision

I would like to review some aspects of the retina, the lateral geniculate body, and the visual cortex and the pathways that connect them. The basic descriptions of the structure of the visual system found in classical writings have guided the efforts of physiologists and psychologists in planning and interpreting experiments on visual function. I will comment on some of these classical teachings in the light of recent experiments in our own and other laboratories.

Let us first look at an example of a typical vertebrate eye, that of a rhesus monkey. We know from psychophysical studies, such as those of Blough and Schrier,[1,12] that monkey vision is quite similar to our own; hence, we should be able to understand a good deal about the human eye from studies on monkeys.

Figure 1 is a low-power picture of the monkey eye, showing the typical results of histologic procedures for fixation, embedding, and staining of eyes. The figure also illustrates one of the problems encountered by retinal histologists: the retina often is detached in fixation or embedding, most often at the fovea. Figure 2 is a higher-power view of the monkey retina, centered at the fovea. Note that in the fovea the outer cellular layers of the retina are displaced away from the dense array of thin central cones. A short distance away from the fovea, the cones become thicker, and they are less numerous and less densely packed.

MORPHOLOGIC DIFFERENCES

Monkeys and man have a mixed retina, in which there are both rod and cone receptors. Many animals have almost exclusively one or the other type of receptor. I would like to discuss some aspects of comparative anatomy of the retina of mammals—both because it is interesting in itself and because comparative study can show in a simple way something about our human retina. Figure 3 is a photograph of the receptors in the retina of a tree shrew, *Tupaia glis.* This relatively simple retina has a single row of cones arranged in a homogeneous mosaic at the back of the eye.[11] There does not appear to be a fovea or any obvious center of specialization within the eye. Such a pure cone retina is a form of spe-

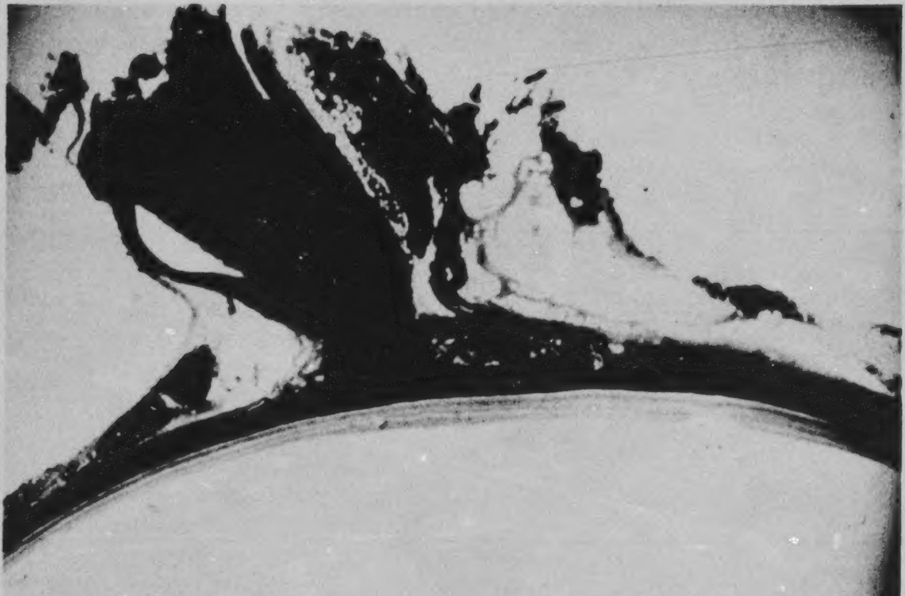

FIGURE 1 Low-power photomicrograph of monkey (*Macaca mulatta*) eye. Susa fixation; embedded in low-viscosity nitrocellulose; Cason's Mallory stain; 10-μ section.

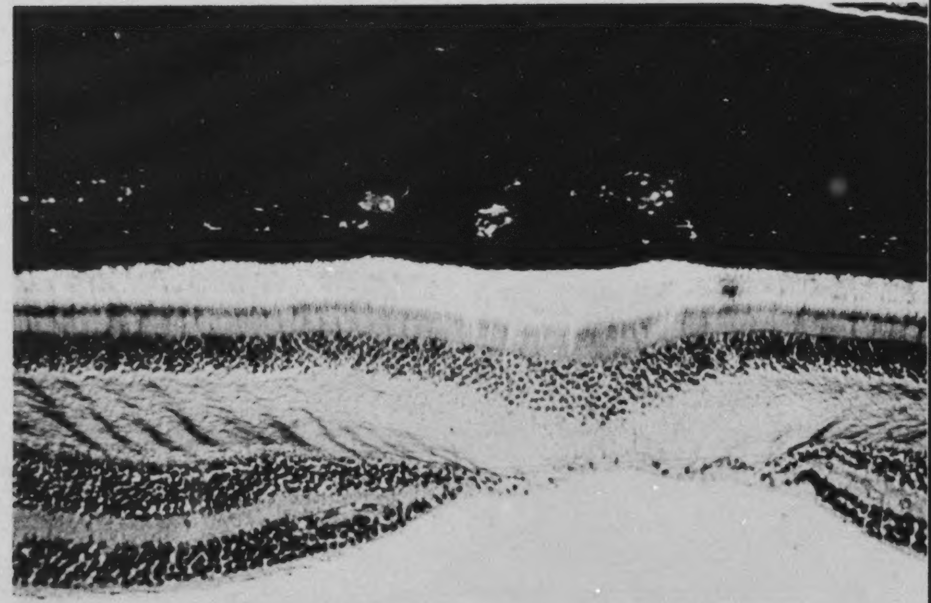

FIGURE 2 Higher-power view of monkey fovea; same section as in Figure 1. (about × 150)

cialization that is found in several species of diurnal mammal. Another aspect of specialization for living in daylight illumination is the dense black pigment that surrounds the outer segments of the cones. The pigment absorbs light that does not strike the outer segments of receptors.

Figure 4 shows the receptor layer of a kinkajou, *Potos flavus,* quite a different kind of animal, which is active largely at night. Zookeepers place it in dim red illumination and often reverse the normal light–dark cycle, because in nature these animals behave very little in the daytime. Their receptors are nearly all rods, with layer after layer of rod nuclei packed below the inner segments of the rods. If one were to package these two retinas together, with rods scattered among the cones and rod nuclei arranged beneath cone nuclei, one might expect the organization seen in Figure 5, the retina of a leopard, *Pantherus pardus.* This kind of receptor and outer nuclear layer is typical of the basically nocturnal animals that are also capable of vision in daylight.

Another major difference in structure associated with nocturnal versus diurnal vision is the ratio of receptor nuclei to different cell types in each of the cellular layers of the retina. If we count the number of receptor nuclei and the number of cells in the inner nuclear and ganglion-cell layers of nocturnal mammals, we see that each successive layer contains fewer cells. There is summation from many receptors onto a smaller number of cells in the inner nuclear layer, and summation in turn from inner nuclear cells onto ganglion cells.

By contrast, Figure 6 illustrates the ratios in a tree shrew. There is a single line of cones, and a roughly equivalent number of ganglion cells. In the inner nuclear layer, which lies between the receptors and ganglion cells, there is a much greater number of cells than in either of the other two cellular layers.

As we noted, many animals capable of vision both at night and in the

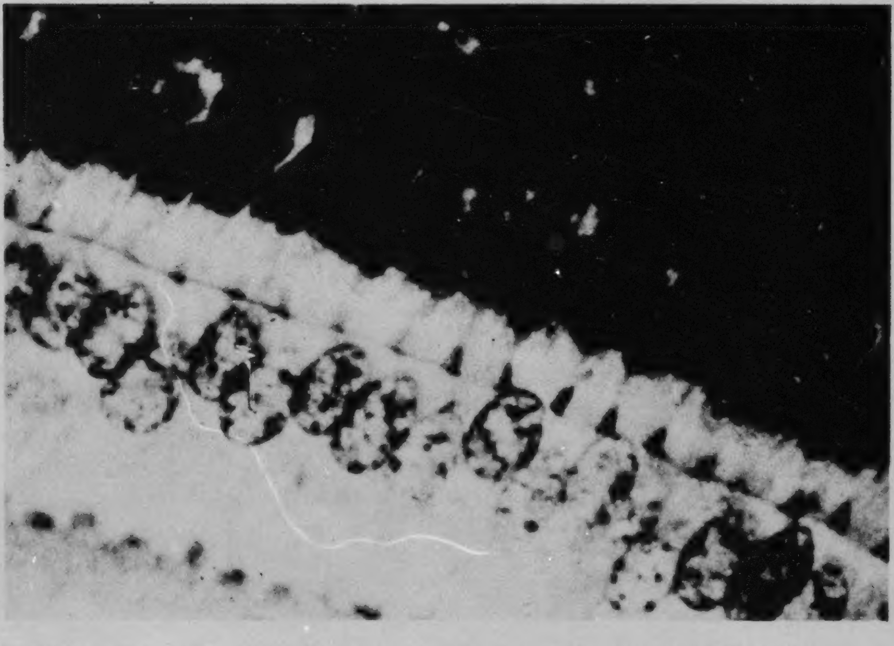

FIGURE 3 Oil-immersion photomicrograph of tree shrew (*Tupaia glis*) retina. Bouin's fixative; embedding, sectioning, and staining as in Figure 1. Note dark pigment layer, part of inner segments of cones, and mottled cone nuclei. (about × 1,540) (Reprinted with permission from Glickstein.[6])

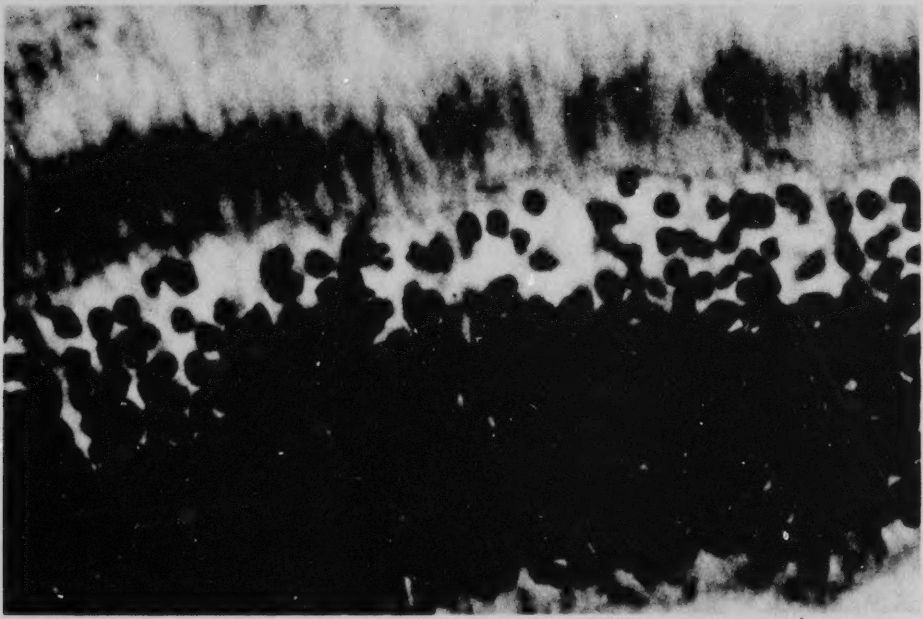

FIGURE 4 Oil-immersion photomicrograph of kinkajou (*Potos flavus*) retina. Histologic techniques as in Figure 1.

daytime have a mixed retina. Figure 6 shows a common arrangement of such retinas: a single line of cone nuclei just adjacent to the outer limiting membrane, below which are many layers of rod nuclei. Figure 7 shows the same general arrangement in the monkey retina a few degrees away from the center of gaze. Note the single line of cone nuclei, and the deeper layer of rod nuclei. This figure shows several identifying characteristics of cone nuclei. Cone nuclei are larger, may stain differently from the nuclei of rods, have a more diffuse distribution of chromatin within the nucleus, and lie closer to the outer limiting membrane.

FUNCTIONAL COMPARISONS

I would like to draw a lesson from some of these structural considerations. The presence of two types of nucleus gives a laminar appearance to the outer nuclear layer. We know that rods and cones operate under vastly different conditions of illumination; hence, there is an easy interpretation of the laminar pattern. The nuclei of cells that function under different lighting conditions are grouped into distinguishable sublayers. Lamination is present in other visual structures, but is not as well understood: the optic tectum of birds and the lateral geniculate nucleus and the cortex of mammals all have a layered appearance.

Figure 8 shows the lateral geniculate nucleus of a squirrel monkey. If one eye is removed and a sufficient amount of time elapses, a covert lamination is revealed in the geniculate.[4] Atrophied cells are smaller than those seen in the lateral geniculate nucleus of a normal animal. The pattern of lamination is such that layers 1, 4, and 6 connect to the contralateral eye, and layers 2, 3, and 5 to the ipsilateral eye. Such an arrangement is present in many old and new world primates, as well as in man.

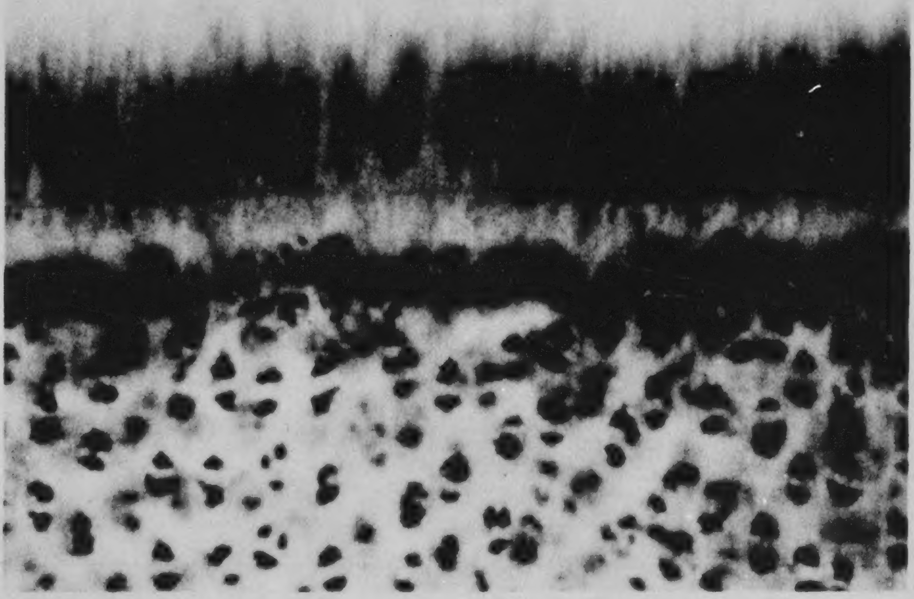

FIGURE 5 Oil-immersion photomicrograph of leopard (*Pantherus pardus*) retina. Histologic techniques as in Figure 1.

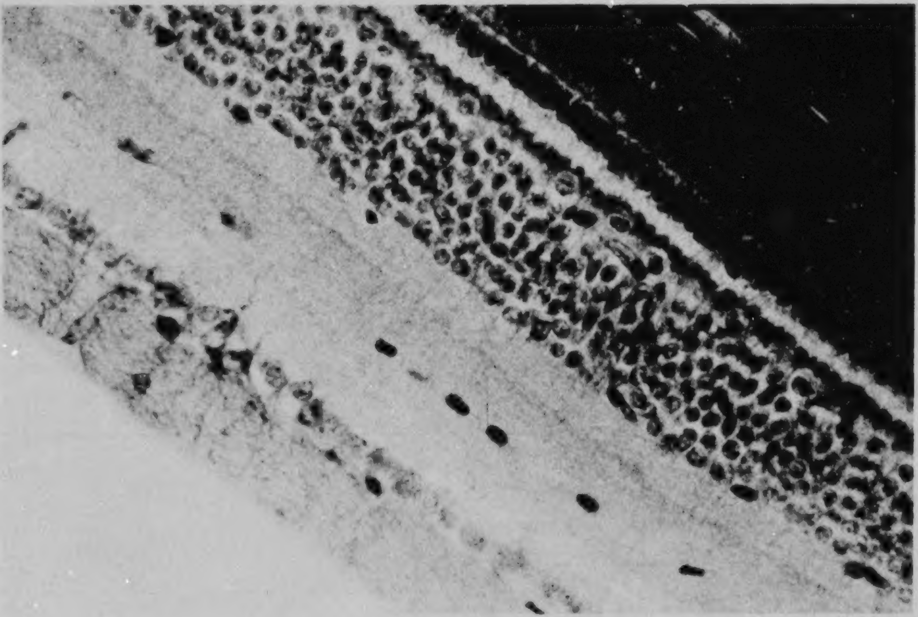

FIGURE 6 Photomicrograph of tree shrew (*Tupaia glis*) retina. Histologic techniques as in Figure 1.

Although geniculate lamination may be obvious, functional interpretation of lamination is not. The visual fields are mapped and remapped six times in the lateral geniculate nucleus of the monkey, and at least three times in the cat. Although a beginning has been made in analysis of receptive-field differences of cells in individual layers of the geniculate,[2,17] there is no clear answer to the question of what is being segregated.

PROJECTION TO CORTEX

I would like now to consider the projections from the lateral geniculate nucleus to the cortex. Classical teachings would hold that there is only one representation of the visual fields in the cortex. The cortex is said to be topologically organized and unique, with neighboring points on the retina projected onto neighboring points on the cortex, and the entire retina represented once and only once.

The physiologic study of the problem of visual cortical projection had its origin in Wade Marshall's laboratory. Talbot and Marshall[14] first began to study systematically the potentials evoked in the cat's brain by flashes of light. On the basis of their observations and those of later workers, we know that gross evoked potentials to flash can be recorded not only in area 17, the striate cortex, but also in area 18. Indeed, Doty[3] showed that the evoked potentials in area 18 are of short latency and actually of higher amplitude than those in area 17. Evoked-potential studies reveal that area 18 also maps the visual fields in an orderly way; hence, Talbot[13] names this region of cortex "visual II." Until recently, it was usually assumed that activity in visual II is due to indirect activation via a relay from primary striate cortex. However, anatomic studies

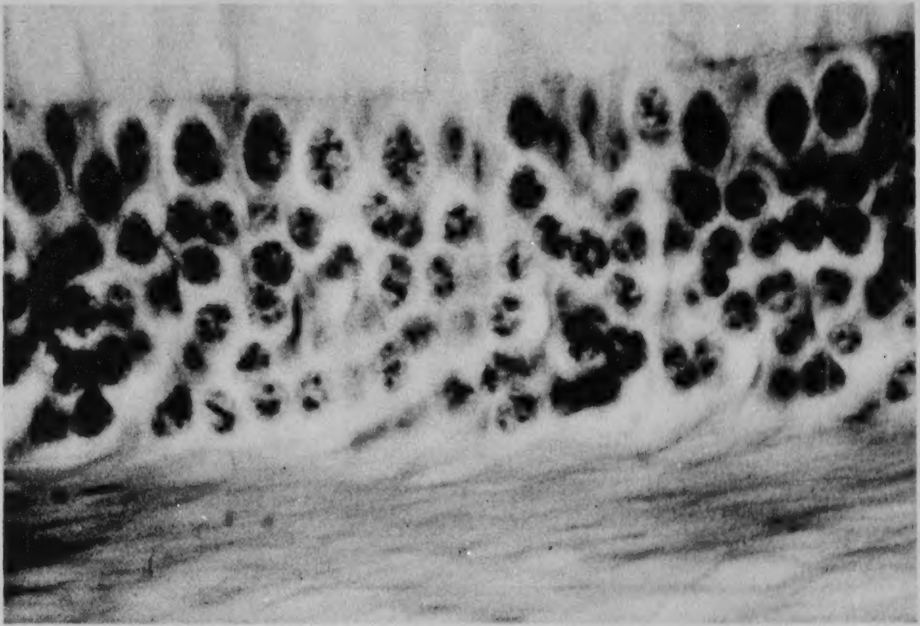

FIGURE 7 Oil-immersion photomicrograph of monkey (*Macaca mulatta*) eye; same section as in Figure 1.

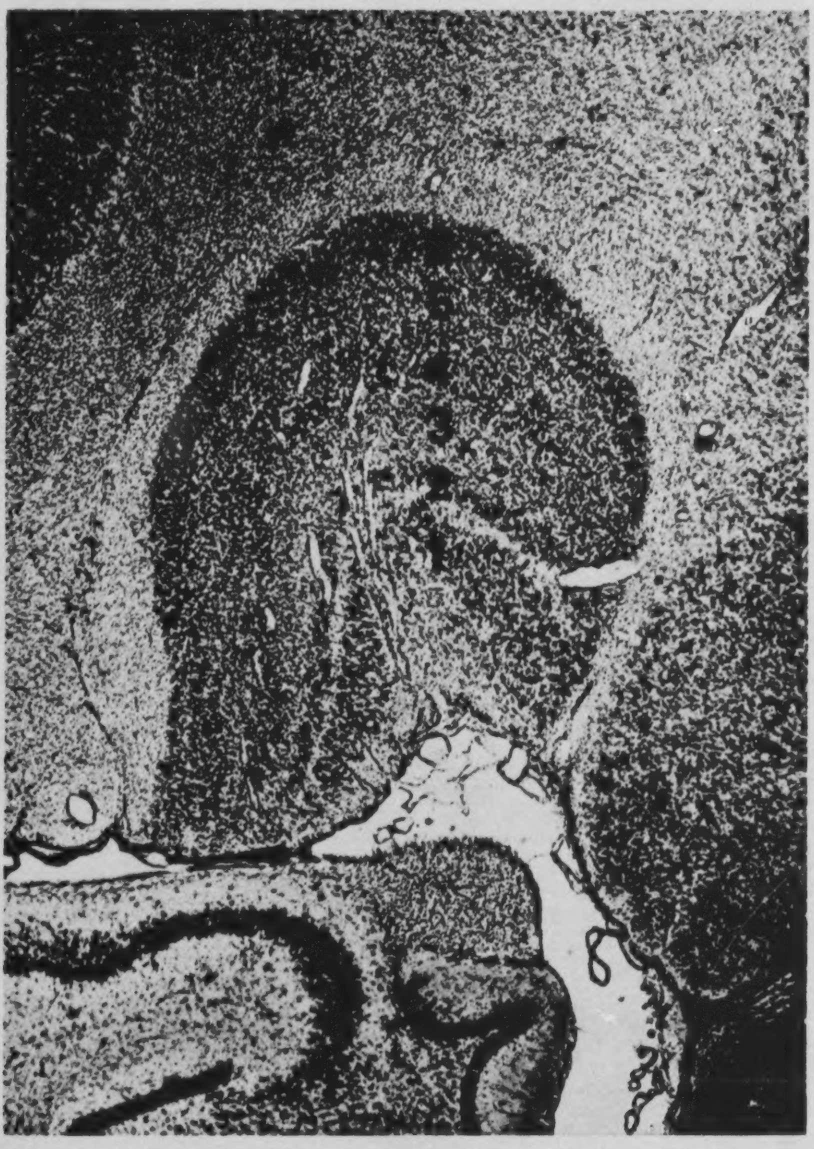

FIGURE 8 Low-power photomicrograph of lateral geniculate nucleus of squirrel monkey (*Saimiri sciureus*) 1 year after enucleation of ipsilateral eye. Note transneuronal atrophy of layers 2, 3, and 5. (about × 13)

reveal that visual II of the cat receives a dense and heavy input of nerve fibers from the geniculate.[7] In the cat, there appear to be at least two ordered projections from lateral geniculate to cortex, each of which maps the visual fields in parallel.

INTERPRETATION

What functional interpretation might there be for parallel projection from the lateral geniculate to two independent regions on the cortex? I would like to raise this question first with relation to theories of the visual function. Classical neurologic thinking is influenced heavily by the concept of a unique cortical projection of the lateral geniculate to area 17. Visual recall is thought to be a function of connections from primary visual cortex to association cortex nearby. Current theories of receptive-field organization,[8,9] for example, suggest that cells in area 18 derive their complex receptive fields from simpler receptive fields of cells in area 17. However, the input from the geniculate to area 18 is a major one in the cat. This projection is both in parallel and in series with area 17. We might try to think of some aspect of vision that might be mediated by area 18. Gordon Walls[16] suggested, and I think it is a good argument, that area 18 of the cat may be a visual center regulating visual reflex actions—reactions of the head and neck, reactions of the eyes—that in the cat serve to maintain objects in constant view.

I do not believe that there is good enough evidence that there are two independent projections of the lateral geniculate in man. I also must confess that I feel that the answer to the problems of the structural basis of developmental dyslexia must be found outside the classical pathways of the visual system. Animals and man can tolerate a surprisingly large loss of the visual cortex without major symptoms of blindness. Lashley[10] showed that rats deprived of striate cortex, although visually impaired, were capable of solving problems based on form if 1/50 of the cells of the visual cortex remained. I confirmed that fully for the monkey. There is also the evidence of Galambos *et al.*,[5] who cut more than 85% of the optic tracts in cats, leaving only a tiny fraction of the visual system, and yet could train form discrimination to a high level.

One might argue that functional use of residual visual cortex occurs only in animals, but there are human cases that show the same thing.

Teuber *et al.*[15] discuss a man who had a massive peripheral scotoma with only a small region of central vision remaining after brain injury. His condition escaped detection during hospitalization; he went to work as a mail sorter, and his disability was not discovered until he thought he needed glasses several years later.

Although the visual pathways are fascinating, I am not sure of their relevance to an understanding of disabilities in reading. The evidence suggests to me that the deficit in reading disorders lies elsewhere.

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DISCUSSION

DR. ALPERN: It is necessary to raise a word of caution regarding the concepts of interaction of rods with cones. The inhibition of rods by cones is frequently proposed as the "explanation" for a variety of psychophysical phenomena, for example, the failure of the rods to play an important role in daylight vision or to determine threshold in the early moments in the dark after a full rhodopsin bleach. Such suggestions are usually made glibly without good evidence, and we now know that the "explanation" is usually wrong. When one looks hard, the almost invariable finding is that rods, far from being inhibited by cones, are inhibited by other rods, and by other rods alone (Alpern, J. Physiol. 176:462–472, 1965). There are, however, three phenomena known to me in which hypothesized inhibition of rods by cones cannot yet be dismissed: (1) in the pupil reflex to light (ten Doesschate and Alpern, J. Neurophysiol. 30:571, 1967, Figure 7); (2) in the color matches of the extrafoveal retina (Clark, Optica Acta 7:355–384, 1960); and (3) in the occlusion of action potential spikes of the ganglion cells in the monkey retina (Gouras and Link, J. Physiol. 184:499, 1966).

Because we can functionally measure differences in the contributions of rods and cones, at the least, by their different spectral sensitivities, directional sensitivities, and kinetics of their visual pigments, it is not unreasonable to expect proposed cone inhibition of rods to be documented in at least these three ways. This has never been done.

DR. GLICKSTEIN: Cone signals can pre-empt the ganglion cell.

DR. MARSHALL: You question the retrograde connections of area 18 to the lateral geniculate nucleus.

DR. DOTY: I did not find any degeneration after taking out this high-amplitude strip. There was a slight amount left that might have damaged fibers going into area 17. The amount of retrograde degeneration in man involves degeneration from area 18, occurring in a little nuclear group abutting the lateral geniculate nucleus on its median dorsal edge. The cat has a little extra area on the geniculate—that is, on area 17, far anterior on the marginal gyrus.

DR. GLICKSTEIN: There is a recent paper by Garey and Powell [Proc. Roy. Soc. (Biol.) 169:107–126, 1967] in which retrograde degeneration was found in the lateral geniculate after lesions were placed in area 18, so I think the retrograde studies are going to confirm the antegrade studies.

DR. DOTY: I can confirm that. I have found that area 18 was just as full of degenerating material as area 17 was.

DR. GLICKSTEIN: There is a little dot in the corner of one of Dr. Marshall's illustrations (J. Neurophysiol. 6:1–15, 1943). It has a short-latency evoked potential to flash similar to that in 17 or 18. This region also receives a direct lateral geniculate projection.

DR. BERING: Are you referring to efferent fibers to the retina from the cortex?

DR. GLICKSTEIN: Efferents to the retina from cortex do not exist. However, in birds, there is a demonstrated efferent projection to the retina from the isthmo-optic nucleus [Cowan and Powell, Proc. Roy. Soc. (Biol.) 158:232–252, 1963]. It is a small nucleus just medial and deep to the tectum, which seems to send a definite efferent projection out of the brain. Brindley and Hamasaki (J. Physiol. 184:444–449, 1966) presented histologic evidence against the view that the optic nerve of the cat contains efferent fibers.

DR. MARSHALL: MacLean and associates (J. Neurophysiol. 31:870–883, 1968; R. Hassler and H. Stephan, Eds., *Evolution of the Forebrain,* 1966, pp. 443–453) found that Myers's temporal loop projects visual signals into the posterior hippocampal gyrus, and certainly gives a direct entrance into a system having an influence on the emotional functions, i.e., the limbic system. The evidence of that is microelectrode findings of photically responsive units in the posterior parahippocampal cortex and the results of neuroanatomic studies, using a recent modification of the Nauta-Gygax technique for demonstrating fine cortical fibers.

ROBERT W. DOTY

Modulation of Visual Input by Brain-Stem Systems

SOME DIFFICULTIES WITH THE TOPOLOGIC MATRIX

The brain creates the world of visual experience from 2 million unit-pulsed fibers in the optic nerves and does it by processing this mosaic of digital input into higher order abstractions that are smoothly continuous in space and time. The manner in which this is accomplished is still so far from adequate scientific explanation that the description "miraculous" is appropriate. Efforts to comprehend this process have emphasized that the retinal projection maps the world on the cortex. Recent elegant elaborations of this approach have demonstrated feature extraction and a hierarchic organization within this topologic matrix (e.g., see Hubel and Wiesel[14]). Although these phenomena are unquestionably of great importance and relevance, a number of recent observations cannot be incorporated readily into this basically topologic approach to the explanation of visual phenomena.

The most disturbing observation is that the topologic system in the neocortex of cats (areas 17 and 18) can be fully removed without apparent detriment to such complex phenomena as pattern vision and visual estimation of distance, provided the removal takes place in the neonatal period.[6] The same appears to be true in tree shrews, even as adults,[17] and possibly to a lesser degree in adult cats.[19] Thus, the exquisitely refined neural circuitry of the topologic matrix has no necessary relevance to pattern vision, although it would be difficult to believe that the systems of areas 17 and 18, when present, do not function in such phenomena. It is also unlikely that higher primates could perform pattern and distance analyses under any circumstances in the absence of area 17; but that does not lessen the problem of defining the necessary attributes of a system that can function well in cats in the absence of the cortical topologic system.

There is now evidence that the input can be scrambled by random destruction of 95% of the optic tracts in cats, and yet maintain some degree of pattern discrimination and a normal distribution of photically evoked potentials.[12,13] That suggests that the punctate information optically focused at the retina is widely elaborated, both at the retina and in the visual cortex. Further electrophysiologic evidence of such elaboration is seen with localized electrical stimulation of the retina: weak stimuli applied even to the nasal retina can evoke potentials in the ipsilateral visual cortex that somewhat resemble photically elicited potentials.[8] In addition to this extensive elaboration of the signal within the visual system far beyond the confines of point-to-point projection, there is wide distribution of photically elicited potentials within the neocortex in both primates[1] and cats (see the contribution of Buser, p. 157). In cats, many of these areas of neocortex remain responsive to photic stimulation even after total extirpation of visual cortex and degeneration of the lateral geniculate nucleus, pars dorsalis.[7]

Perhaps equally disturbing to any simplistic concept that the visual system operates via a mere topologic hierarchically organized matrix is the fact that visual information can be drastically modified at the first and subsequent central relays by action of the centrencephalic system. In primates, this gating function seems to be exerted predominantly at the lateral geniculate nucleus. This influence is so powerful as to suggest that it constitutes the *raison d'être* for this thalamic relay nucleus.

Because some features of the electrophysiology of the visual system of primates differ importantly from those in cats,[10] I will present a brief summary of some of them before proceeding with discussion of the centrencephalic influences.

GENERAL FEATURES OF THE ELECTROPHYSIOLOGY OF THE PRIMATE VISUAL SYSTEM

Whereas in the cat it is relatively easy to elicit potentials throughout areas 17 and 18 by stimulation at a single electrode placement in the optic tract, it is essentially impossible in squirrel monkeys and macaques. Apparently, the glial investiture of fiber bundles in the primate optic tract has such a high electrical impedance that effective current spread is severely limited, and only a small group of fibers can be excited from any one placement. Surgical levels of anesthesia severely depress synaptic transmission at the lateral geniculate nucleus (LGN), and even one-tenth the anesthetic dose of Nembutal lengthens the recovery time of synaptic transmission from 15 msec to 50–100 msec. These facts impose a number of technical difficulties in exploring the electrophysiology of the central visual system in primates.

In the primate LGN, there is a clear separation, of unknown importance, into magnocellular and parvocellular laminae. The large cells in the LGN are innervated by the fast fibers from the retina, and photically elicited potentials occur about 5 msec earlier in magnocellular than in parvocellular laminae.[5] Conduction velocities of the fast and slow systems are 15 and 6 m/sec, respectively in the optic tract and at least twice as high in the optic radiation. Paradoxically, the parvocellular elements recover synaptic transmission after an excitatory volley slightly faster than do the magnocellular elements—about 8 msec versus 12 msec to full recovery in the unanesthetized macaque with chronically implanted electrodes. The first cortical synapses recover even faster. The cortical response to a single volley ascending the optic radiation is similar to that in the cat, except that there are apparently twice as many waves because of the separation in time of arrival at the cortex of the magnocellular and the parvocellular components. Electrophysiologic and anatomic evidence suggests that the magnocellular group does not project to the foveal representation in the area striata, but this needs further investigation.

One of the more gratifying things in working with macaques with permanently implanted electrodes in their visual systems is the realization that evoked potentials are commonplace and are not just a creation of the artificial conditions of electrophysiologic experiments. As the animal looks about in a normally lighted room, potentials, complete with high-frequency oscillations,[9] are constantly being evoked in optic tract and striate cortex by changes in direction of gaze.

CONTROL OF EXCITABILITY IN THE CENTRAL VISUAL SYSTEM IN PRIMATES

In most squirrel monkeys and macaques, the excitability of the striate cortex to a volley ascending the optic radiation is greatly reduced if the unanesthetized animal is in the dark. Thus, some background activity from the retina has a very important role in controlling cortical excitability. For unknown reasons, the effect of this activity (the Chang effect) in some animals is minimal, and in most it does not change transmission at the LGN. Bilateral enucleation, however, has very dramatic effects at both the geniculate and the cortical levels. The time course of the changes after enucleation has not been carefully studied, but the changes are well developed within a few hours, and over the course of 2 to 3 days they reach a maximum that is maintained indefinitely. In one animal, a "world record" evoked potential was obtained: the responses in area 17 to stimulation of the optic tract changed from 100 μV peak-to-peak preoperatively to as much as 9 mV after enucleation. This great change in excitability to afferent excitation is accompanied by the development of a very bizarre, convulsive type of electroencephalogram (EEG) in area striata, with 0.5- to 2-sec runs of high-voltage irregular spikes, punctuated by approximately equal periods of almost complete silence. Similar bizarre patterns have been recorded in the EEG of the human blind.[4,15] It is thus apparent that, in addition to the modulation of central excitability exerted by the retina in light, compared with dark, there is some powerful control of the retina itself over background activity of the central system.

In the normal macaque sitting in the dark while potentials are evoked at different points in area striata several millimeters apart for stimulation of the optic radiation, great independent variation is seen in the excitability of the several striate loci. One gets the impression that each point of the cortical mosaic is subject to a large degree of localized control in the dark. Still more dramatic changes of a more global nature occur with fluctuation in the attentive state of the animal in the dark. Indeed, when the monkey is relaxed and probably dozing, transmission through the LGN almost ceases for single volleys coming over the optic tract, and the cortical response is correspondingly reduced. The reduced cortical response, however, belies the true state of the cortex, in that stimulation of the optic radiation at such times produces a severalfold increase in the cortical response. Thus, as attention lags, the LGN is "shut off" and the cortex "runs loose." Other data from studies of evoked potentials, as well as data from enucleation studies, similarly suggest that activity in the LGN somehow has a tonically inhibitory influence on area striata.

When the animal is alerted, the foregoing comparison of excitability at the cortex and the LGN is immediately reversed. The fluctuation in excitability at the LGN can be very rapid. For example, in one squirrel monkey, as the optic tract was tetanized at 30 pulses/sec, it could be seen that the response in the optic radiation often varied severalfold from one pulse to the next.

In acute experiments with light barbiturate anesthesia, it is readily shown that the focus of this system that modulates LGN excitability is in the mesencephalic reticular formation (MRF). A single pulse applied to the MRF shuts off transmission through the LGN within about 8 msec and keeps it suppressed for 25–30 msec. Recovery is complete by about 50 msec, and it is usually followed by great augmentation of the response for about 100 msec. Studies show that the inhibition so obtained is presynaptic.[16] However, the facilitory effect is prepotent, and in unanesthetized monkeys the inhibitory effect disappears (Wilson, Pecci-Saavedra, and Doty, unpublished data). Also, in some anesthetized preparations it is difficult to obtain the inhibitory effect, whereas the facilitory effect may always be obtained unless the animal is already at a peak of alertness (as is often true with unanesthetized macaques). When the inhibitory effect is present, it is overwhelmed by facilitation if a short train of three to six pulses at 300/sec, rather than a single pulse, is applied to the MRF. Similar facilitation can be obtained by pulse trains applied to the superior colliculus, vestibular nuclei, locus ceruleus, and other areas; but it is never as great, and the threshold is always considerably higher than for stimulation of the MRF. Facilitation of the LGN is unaffected by enucleation or removal of the area striata. Unlike similar effects in the cat, facilitation can be observed in photically elicited responses, as well as in responses to an electrically elicited volley.

The pathway by which these facilitory influences reach the LGN from the MRF is still obscure and is probably diffuse. It does not seem to follow the brachium of the superior colliculus, and it survives extensive brain-stem lesions in the area between the two structures.

The significance of this control of visual input is equally obscure. P. O. Bishop (personal communication) has made the ingenious suggestion that it might be related to control of inputs lying within and outside the horopter. However, at least some aspects of the control are probably related to eye movements. This is suggested by the extremely rapid fluctuations in LGN excitability in normal squirrel monkeys, as noted, and by the occurrence of potentials in the parvocellular portions of the LGN 50–70 msec after the occurrence of eye movements.[11] Facilitation does not occur after stimulation of the oculomotor nucleus; hence, if it is associated with eye movements, it does not arise as a direct feedback. The MRF stimulation that facilitates transmission through the LGN is not consistently linked to loci controlling eye movements, and the potentials that appear in LGN in response to eye movements also occur in response to tactile or auditory stimulation.[11] In cats, potentials in the LGN also occur during eye movements and are correlated with discharge in the pons,[3] discharges of neurons in the visual cortex,[18] and presynaptic inhibition of the LGN.[2]

To gain some insight into the meaning of the modulation of LGN excitability by the MRF, John Bartlett in my laboratory has been studying the effect of MRF stimulation on responses of single units in the area striata of painlessly immobilized, unanesthetized squirrel monkeys. A natural stimulus, such as a moving line, is presented, to which the unit responds; 50 msec before every other presentation, the MRF is stimulated with six pulses, which in the nonimmobilized animal produces a mild alerting. The average of 10–15 presentations with and without the MRF stimulation shows that the unit responds faster when the visual input is preceded by the alerting stimulus. So far in these still preliminary experiments, this effect holds for all classes of units (e.g., those responding to intensity of diffuse illumination, to movement, and to various combinations of color and movement), and seems merely to mimic the change in response obtained by increasing the intensity of the natural stimulus. Perhaps with further analysis, some difference in pattern of discharge may be discernible for change in intensity versus change in alertness, but it is not yet apparent.

A major problem thus arises concerning the ambiguity of the information passed on by the LGN, which may not be an accurate reflection of the event in the real world. The ambiguity is, of course, compounded by complex interactions among abstracted qualities of the stimulus. For example, some of the units found by Bartlett display directional sensitivity that is color-dependent. Thus, with white light, a unit may respond best when a line is moved toward, say, 1 o'clock, but respond still more vigorously to a red line whose intensity is about 1 log unit lower moved in the same direction. When the line is green, however, the direction of movement giving the same maximal response changes to 3 o'clock. Thus, discharge of this particular unit may not distinguish between red and green lines moving in different directions, these lines moving in nonoptimal directions but preceded by MRF stimulation, or a white line moving in the optimal direction after MRF stimulation. In other words, discharge of this unit is ambiguously compounded from color, direction, velocity, intensity, and state of alertness. How, from such a mélange, the nervous system can form a representation of reality is obviously still elusively miraculous. However, from such complications it can at least be inferred that the process significantly transcends a mere duplication and hierarchic extraction of the retinal image in the topologic matrix of the cerebral cortex.

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ELWIN MARG

A Neurologic Approach to Perceptual Problems

Dr. Boynton has suggested that we might learn something more about perceptual problems by exploring the receptive-field organization of visual neurons in the human brain. This has been our goal for almost a decade, and we have developed methods for doing it in man. These methods are based on the use of microelectrodes developed for implantation in the brains of patients with intractable temporal lobe epilepsy.[7,9] The patients in our study were in a group studied by Dr. John E. Adams of the University of California Medical Center in San Francisco. They were to undergo diagnostic and therapeutic brain surgery for relief of their seizures and consented to having the fine microelectrodes added to the usual gross ones.[8,10]

Briefly, the method involves implantation of flexible bundles of eight microelectrodes (Figure 1) in the cortex. Each electrode is made from a 50-μ straight tungsten wire etched to a 1-μ tip and coated with multiple layers of Isonel 31. They may be included in an indwelling microdrive that can move them from one neural unit to another in the cortex, or they may be left in a fixed cortical locus, in which case electrical pickup of single units is likely because of the large number of active neurons at the tip (Figure 2).[5] The electrodes are introduced through a burrhole 2 cm to one side of the inion. All we can say in identifying

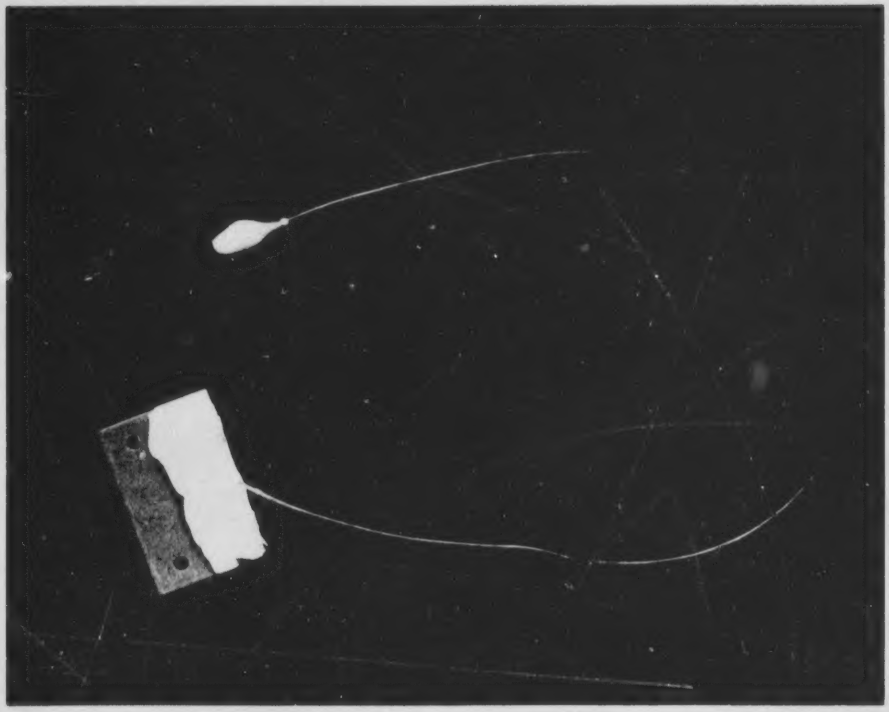

FIGURE 1 Microelectrode bundle consisting of eight microtips loosely held together by a small segment of plastic tubing. A separate "ground" lead is deflected to one side. In this model, the tungsten wires are welded to insulated stainless-steel leads for greater flexibility and length. The splice is within the Silastic mass, which is held firmly in the burrhole by the application of additional Silastic, which forms a plug.

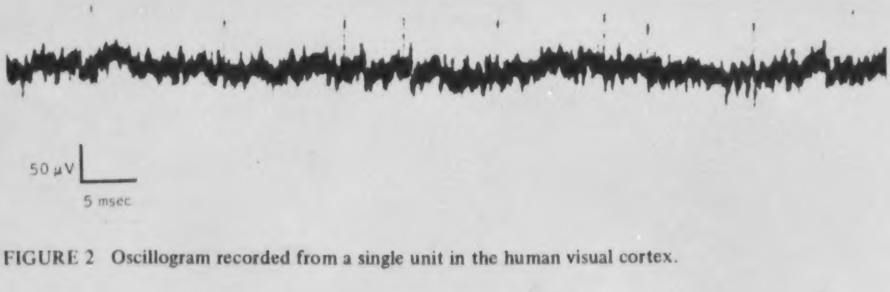

FIGURE 2 Oscillogram recorded from a single unit in the human visual cortex.

the cytoarchitectonic areas is that they are in the visual cortex. It is impossible to distinguish between areas 17, 18, and 19 without histologic confirmation, which we have never had.

Dr. Richard Jung and co-workers[6] first recorded single units in the visual cortex of experimental animals; Hubel and Wiesel[5] and others later demonstrated the receptive-field organization of these cells. The human cortical receptive fields resemble, with some important differences,[8,10] those found in the monkey.[4]

In a series of 15 patients, we observed many units in the visual cortex that did not seem to respond to any stimulus we could provide, whether visual or otherwise. Their "bursty," spontaneous activity appeared independent of external influences. Other units showed a response superimposed on the spontaneous activity when targets were brought within the visual field. This electrical response was amplified until it could be heard over a loudspeaker. With this type of response, we were able to plot nine receptive fields, five in response to disks and the others to bars or lines (Figure 3). The patient fixated a mark on a large sheet of cardboard 1 meter from his eyes. We then moved bars and disks of various sizes and colors and mounted on stiff wire wands within

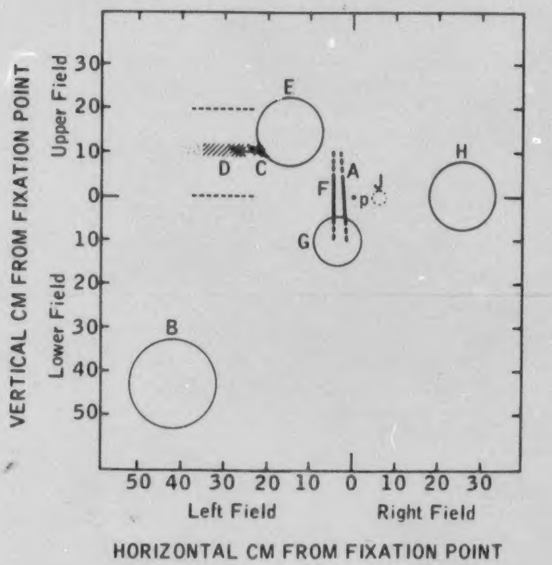

FIGURE 3 Receptive fields recorded from single units in the human visual cortex. See text for explanation.

his field of vision and listened for a response. The receptive fields were outlined in pencil on the cardboard for later measurement.

Because plotting was rapid and could be repeated rapidly, and because the edges of most of the receptive fields were sharp, any wandering of fixation could be detected and thus did not affect the size or position of the plot. Generally, the patients were very cooperative and maintained visual fixation well. Monocular and binocular fields were plotted, and we detected in these patients the various degrees of dominance that have been reported in laboratory animals.

All the receptive fields that we plotted had some characteristics in common. The responses to black on a white background, white on a black or red background, and red, yellow, green, or blue on any contrasting background were equal: the cells were, so to speak, all color-blind.

None of the units or their plotted receptive fields could be influenced by a patient's efforts to change them. For example, we increased the audio gain until the patient could hear the pulses of a unit firing in his cortex and then asked: "Can you hear that? Can you do anything to influence it? Can you increase or decrease it, or affect it in any way?" No matter how much the patient tried to influence the response, we could detect no changes. We also brought the target into the receptive field and asked: "Did you hear that sound when the target was brought here? Now, the target is withdrawn. Imagine it is there and try to make the same sound come from the loudspeaker." No one succeeded in doing that.

None of the units appeared to be influenced by stimuli to other sensory modalities.

All the plotted fields came from excitatory or "on" units, the response being superimposed on the irregular, "bursty" spontaneous rhythm. If there are any inhibitory or "off" units, they appear to be uncommon.

A single unit was usually recordable for the length of a 1- to 2-hr session. At times, a unit would be recordable from one day to the next over the same microelectrode.

The maximal receptive-field response occurred when the target stimulus matched the size and shape of the field. This simple method of target presentation would not be expected to elicit the response of a weak inhibitory surround, and, in fact, we could find no evidence of such a response.

One aspect of our work has direct bearing on the plasticity of the brain, a subject basic to the interests of this conference. Some units showed a progressive attenuation[3] or habituation[2] in their response to repeated stimulation in their receptive fields, which lasted longer than the minute or two reported for laboratory animals. The phenomenon was cortical, in that habituation of a binocular unit by the stimulation of the receptive field of one eye caused a decrement of response in the receptive field of the other eye. A new, nonmonotonous stimulus, either to vision or to another sensory modality, did not restore the response–i.e., did not produce a dishabituation.[1]

The receptive fields are plotted in Figure 3, in which "p" is the fixation point 1 meter from the eyes. "A" is a receptive field (width, 17 min of arc) of a unit in the ipsilateral or left cortex; the left eye was dominant. All other receptive fields were contralateral to the cortex where their units lay. "B" is a monocular field, right eye, as is "C." "D" is a complex receptive field. Here, a horizontal bar would give a response anywhere within the extent of the field delineated by the horizontal dashed lines. This receptive field also showed marked habituation. "E" is a disk-shaped and "F" is a bar-shaped binocularly equal receptive field; "F" showed marked habituation. "G," "H," and "J" are binocularly equal receptive fields; only "G" showed strong habituation.

Spatial plasticity was also observed in some receptive fields. There appears to be a systematic change with fixation distance of some receptive fields of the angular diameter and the angular position relative to the fixation point. It may involve a size constancy or scaling mechanism.[11,12]

If we are going to investigate plasticity and other subtle functions of the brain, we should, for several reasons, do it in man. First, there appear to be species differences, even between man and monkeys, in receptive-field organization. Second, man not only cooperates with prolonged fixation and specific directed eye movements, but can also be directed to make mental efforts and to describe perceptual responses to stimulation. In this way, the relationship, in terms of unit activity, between simple and complex perceptual tasks, such as reading and its neural basis, can be investigated.

Future developments in neurosurgery may increase the number of potential volunteers for these perceptual-neurophysiologic studies by making unit recording valuable in prognosis and diagnosis of postoperative patients with evacuated hematoma or traumatic encephalopathy. Indwelling microelectrodes that probe the neural organization of the brain should shed more light on the neurophysiologic basis of perceptual disorders.

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PIERRE BUSER

Nonspecific Visual Projections

In an interdisciplinary discussion of dyslexia, two categories of electrophysiologic data on the visual system of animal and man may be of interest. One category concerns what we consider the primary visual system, from the retina to the cortical receptive area. There is no doubt that increased knowledge of the organization and functioning of this pathway, considered at the retinal, thalamic, cortical, and collicular levels, is essential to clarify some of the major problems posed by dyslexia (see the reports by Glickstein, p. 130, and Doty, p. 143). The other category includes data related, in one way or another, to other visual projections in the brain. The study of the primary system does not encompass all that we know about the spread of visual information through the cortex or subcortical structures. Animal experiments have clearly indicated for more than 20 years that visual projections exist in many structures outside the primary pathway. We may call these "nonprimary" or, to use a more common but somewhat misleading term, "nonspecific."

It is precisely when considering higher integrative processes, such as reading, that one must bear in mind the existence of nonspecific projections, especially because many of the "higher functions" (pattern recognition, memorization, association processes, and so on) may depend on these nonspecific projections at cortical or even subcortical levels. Therefore, it seems relevant to summarize very briefly various observations in this field. Although it is necessary to concentrate on functional data, I shall discuss, first, observations on the "topographic" extent of nonspecific projections and, second, results indicating their possible role in higher integrative processes.

EXTENT OF VISUAL PROJECTIONS FROM TOPOGRAPHIC DATA

Electroanatomic studies in the cat, and a few in the monkey, have indicated the existence of visual projections to various stations outside the primary pathway–mesencephalic, diencephalic, and cortical. It is true, however, that the extent of such projections is still a matter of discussion. In fact, their apparent extent depends heavily on experimental conditions, such as physiologic characteristics (e.g., level of attention or wakefulness) when free animals are considered and type of preparation (for instance, anesthetic) when "acute" investigations are performed.

It is beyond the scope of this brief review to consider all these topographic data and discuss the anatomic problems that they raise. But it may be of interest to point out some aspects as an introduction to functional considerations.

At the reticular level of the mesencephalon, the data show visual inputs, with longer latencies and far greater variability than in the primary pathway, including the superior colliculus.[9,19]

Among extrageniculate thalamic nuclei that respond to stimulation of the retina, some belong to the "associative" group, as defined anatomically, such as the lateralis posterior and pulvinar. However, visual responses have also been characterized within another group of thalamic structures, namely those belonging to the group of nonspecific, diffusely projecting nuclei, as defined through physiologic methods by Dempsey and Morison[20] and later by Jasper[31] and others. Anatomically, these nuclei include intralaminar nuclei, midline nuclei, and the reticular nucleus. Here again, the electrical visual activities recorded are much more variable and display longer latencies than those at stations in the primary system.

At the cortical level, systematic studies in cats have revealed a variety of projections to areas outside the primary receptive field, following diffuse or focal illumination of the retina. Some can be recorded from the associative cortices: in the cat, the suprasylvian gyrus, the anterior lateral area, and some areas on the medial aspect[9]; and in the monkey, areas in the frontal lobe[5] and superior temporal gyrus.[27,39] Such responses exist also in the motor cortex, a fact that explains why light stimuli can elicit pyramidal discharges in some experimental conditions. Although there are indications that some of these cortical inputs originate from associative nuclei, most of the cortical extraprimary projections seem to depend on the nonspecific system–reticular and thalamic. The exact natûre of this dependence is unknown. (See Buser and Bignall[9] for discussion.)

Pathways from nonspecific subcortical centers also project on the primary visual system itself at the geniculate level, from the reticular formation,[2,10] and at the cortical level, from various mesencephalic or thalamic sources.[8,16,18]

Finally, almost all nonspecific structures throughout the brain–whether reticular, thalamic, or cortical "associative" and motor area–are multisensory, i.e., they respond almost equally well to visual, acoustic, and somatic sensory inputs.[1,4]

FUNCTIONAL DATA

In general, the functional meaning of the nonspecific projections of visual input is largely hypothetical. Nonspecific visual projections exist in subcortical structures that have already been shown to be essential for regulating states of alertness (wakefulness and sleep). That is especially true of the reticular formation of the brain stem. It thus follows that visual inputs may act on the level of wakefulness and selective attentiveness through these structures. It is also possible that the general level of activity of the nonspecific system may control information transfers in the primary pathway. This may occur through a facilitation of the visual cortex or, electrophysiologically, by addition of a multisynaptic late visual input to the earlier input transmitted through the primary channel. The importance of these various processes, which are not mutually exclusive but complementary, remains to be determined.

Some nonspecific projections have their end-stations in cortical areas that have been suggested or shown to be essential to cognitive integration processes. It is hypothesized that visual nonspecific information contributes to such processes of visual association and elaboration. From older theoretical schemes on complex brain operations (as initially suggested by Flechsig[24]), one would expect associative thalamic nuclei to play the major role in conveying visual input to associative cortex. But that does not seem to be the case, inasmuch as the major input comes from nonspecific nuclei. The fact that most of the nonspecific areas are basically multimodal may be important, because intermodality associations constitute one of the prerequisites for cognitive or decision mechanisms.

Let us now consider some facts relevant to these functional problems.

Modulatory Influence

A large group of experiments were undertaken to demonstrate the "modulatory" influence of projections from the nonspecific system (as elicited by electrical stimulation of its central components). Various macroelectrode[7,11,23] and microelectrode[16–18,33,34] studies have shown that stimulation of the reticular formation or of thalamic intralaminar nuclei can modify the pattern of cortical visual responsiveness. Depending on the experimental procedure, these modulatory influences act in various ways, although they often are facilitating rather than inhibiting. This is the case with the visual evoked potential recorded through macroelectrodes and at the single-cell level; thus the temporal discrimination of cells in the visual cortex of cat has been shown to increase during stimulation of the nonspecific system.[34]

Participation in Input

Another category of data emphasizes the participation of the nonspecific system in visual input to the visual cortex. Many studies, performed on animal and human subjects (in the latter case with averaging autocorrelation techniques), have suggested, or even established, that the gross evoked potential due to diffuse illumination of the retina comprises components that are of "nonspecific" origin.[9,12–15,35] These components usually appear slower and later than the others, which represent the activity of the primary input to the cortex or of the cortical neurons that are directly activated. It is also remarkable that these late, slow components (whatever their number and shape) are far more sensitive to various external or internal pharmacologic or physiologic factors, and that (especially in human studies) their cortical localization goes beyond the limits of the visual area.

Correlation with Attention

A third group of data are related to possible correlations between the amplitude of the cortical evoked response to visual stimulation and some psychologic factors, especially variations in selective attention between visual and nonvisual stimuli. Many earlier data were somewhat controversial, mainly because the experimental conditions were not defined with sufficient precision as to the direction and selectivity of the attentive state; recent experiments are far clearer in their conclusions.[6,21,22,25,28–30,32,37] To summarize these data, which were usually (but not always) obtained from scalp recordings in human subjects, it can be noted that gross evoked responses to light (1) tend to decrease when stimuli are monotonously repeated, i.e., when a decline in visual attentiveness and habituation to the stimulus occurs; (2) decrease when a subject is presented with nonvisual stimuli, i.e., when his attention shifts toward other sensory modalities; (3) are correlated with reaction time to visual stimuli, faster reactions being associated with larger responses; and (4) increase when a subject is visually attentive—not only visually searching, but also perceiving significant stimuli.

Using a vigilance task, Haider *et al.*[28] could force subjects into one or another type of attention, visual or auditory. It appeared that, when a subject's attention was directed toward visual and not auditory stimuli, visual responses increased, whereas the sound-evoked potential in the temporal lobe not only declined, but also developed a longer latency. The most conspicuous changes in these experiments involved the late components (peak latencies, 160–300 msec), i.e., those which originate from or depend for their amplitude on the nonspecific system. Such results clearly emphasize the essential role of the nonspecific system in higher elaboration and cognition of visual information at subcortical or, more probably, cortical stations of the visual primary system.

FUNCTIONAL CORRELATIONS

The last group of investigations to be dealt with herein should concern the functional role of visual projections to nonvisual cortex—i.e., to motor or associative areas or even areas that are primary for other modalities. Actually, there are very few data, if any, regarding this subject. In spite of evidence, from ablation studies in animals or clinical investigations in man, of impairment in visual discrimination tasks or visual cognition after lesions of some associative areas, no consistent electrophysiologic findings seem to do more than show the existence of such projections. Except for the fact that evoked responses recorded from the associative or motor cortices in animals display larger variations than those recorded from primary areas under various psychologic conditions (arousal, habituation, and so on), true functional correlations remain to be established.[9] In the monkey, for example, visual responses have been identified, as mentioned earlier, in the parietal–temporal–occipital area, where lesions produce major impairment of visual tasks.[36] Tentative studies of electrophysiologic correlations in this field, during visual discrimination,[26] have failed. Thus, more data are required if we are to understand the function of these associative projections.

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DISCUSSION

DR. ALPERN: In regard to that flash-evoked response in the experimental eye fields, what is wrong about the view that this represents projection along the primary pathways to the occipital cortex and then to some associated occipital pathway? Would that not be the same way of looking at that?

DR. BUSER: Yes, of course, but as long as that has not been demonstrated, my personal bias would favor a parallel system more than a system in series with the visual cortex. That is based on my experience with cats.

Now, can we jump from a cat to man? I do not know. That is why we have to be cautious; I am not ready to jump to that conclusion. There may be another explanation of this in view of Bignall's findings.

DR. LINDSLEY: What about the effect of blinking on these responses? Does this effect happen the moment you get the pathway?

DR. BUSER: No, this is not blinking, I am sure. Blinking would be a micromovement, and in that case it would not be visual information.

DR. GAARDER: If you were to push your finding to the extreme, you might say that an eye jump is also an evoked response. You might say that the cortex is like a giant screen, on every part of which there is some manifestation of a response or an increment of visual input. Then, to speculate further, perhaps the input from one's sensory system is manifested in some way on almost every part of his cortex. This is going beyond anything we can see.

DR. BUSER: I do not think I will go that far, because to me there is a great difference between input and activity. We had some cortices that seemed to behave electrophysiologically like associative cortex, and primary cortices that behaved in quite a different way, even electrophysiologically. The difference between input and activity mentioned earlier is chiefly, I believe, in the nonprimary area. As soon as you enter the classical domain—a primary system—things change completely. Even evoked responses of single units in visual areas—for example, to acoustic stimulation—are different. I do not want to say that the whole cortex has equal potential.

DR. LINDSLEY: Every single muscle is connected by pathways, if you are willing to pursue this deep enough and take the connection to its logical extreme.

DR. BUSER: Yes, but you forget about the long connection pathways. We do not know what these connections do when they arrive upstairs: they are projecting something down. We do not know much, if anything, about the input-output function at the corticocortical level. I am aware that this kind of study is completely artificial: First, we are eliminating the entire retina, and that is probably not the best way to study visual functions; and second, I did not show macrorecording activity, because in that case I would be showing mostly older material.

We are intending to look further for the maximal possible spread of information, and I ended by saying that, according to our data, some of the spread must be modulated by primary areas.

DR. RIESEN: What you have said sounds a great deal like the classical views of Karl Lashley. I wonder whether you have any comment on recent reports by MacLean *et al.* (J. Neurophysiol. 31:870–883, 1968) that he is getting visually evoked activities in monkeys.

DR. BUSER: Our work was in the cat and involved only the lateral convexity on the medial wall. It is perfectly clear that we have the same sort of projections as his.

DR. INGRAM: In what experimental animal situation do you get spread of evoked potentials into the so-called associative areas?

DR. BUSER: I showed the mostly artificial conditions required for this kind of preparation, but I think some would agree that you can also get this result in normal animals. Some functional variations correlate with the potentials' amplitude, such as the state of wakefulness of the animal, as has been described in the older literature.

The general idea is that the nonprimary responses are much more like primary responses in this area and much less sensitive to behavioral conditions like sleep, wakefulness, and so on. By comparing the single-unit response in the nonprimary central medianum nucleus of the thalamus with that in the lateral geniculate, we can show that the state of sleep or wakefulness of an animal is changing all the time. Of course, when the animal is strongly aroused, it is very difficult to record something outside the primary fields.

ROGER W. SPERRY

Cerebral Dominance in Perception

This presentation will be concerned largely with a review of some recent evidence obtained by Dr. Ronald Saul and me[4] on the effects on visual perception of congenital absence of the corpus callosum in man. The behavioral symptoms seen with congenital absence of the callosum will be compared with those produced by surgical elimination of the callosum and other cerebral commissures. In the latter case, of course, the two hemispheres, which have functioned together for years with the channels for cross-communication intact, must suddenly get along without the accustomed direct lines for cross-talk. With congenital failure, the hemispheres must get along from the very beginning without the normal cross-communication. The increased functional compensation that is achieved in the congenital situation, compared with that after surgery in the fully developed system, will give us some indication of the degree of functional plasticity that exists in the growing and developing brain, beyond that seen in the fully developed brain.

We have been fortunate during the last year in having available for testing and study a patient (S.K.) recently diagnosed from x-ray studies to have complete agenesis of the corpus callosum. We had seen others in the past, but this particular patient, first seen by Dr. William Wright at the Los Angeles County General Hospital, has one of those very rare "asymptomatic" cases; no signs of abnormality were discovered until the age of 19, when headaches developed after an acute attack of hydrocephalus. The patient recovered quickly with treatment and returned to college, where she is currently a sophomore with an average scholastic record (C's and B's).

It was our first thought that, even though no functional symptoms had been evident in ordinary behavior, such symptoms associated with loss of the corpus callosum could probably be demonstrated if we could get her into the laboratory and put her through some of the series of tests for interhemispheric integration with which we had been successful in recent years in demonstrating symptoms in surgical patients with cerebral commissurotomy (Figure 1). Normal subjects perform these tests without difficulty, but a group of patients of Vogel and Bogen who have undergone surgical section of the corpus callosum and anterior commissure either fail completely or show gross impairment.[6,8]

Our patient went through every test without hesitation, performing easily and apparently at normal efficiency task after task that had stopped the surgical patients. I will run through a few examples to illustrate the kinds of functions involved, with emphasis on test performances that involve vision and language. These will help to give an idea of the functional reorganization and compensation of the cerebral mechanisms underlying vision and language that are possible in the still developing and growing brain but not in the fully developed brain. This patient exemplifies the functional plasticity of neural maturation[7] that is presumed basic to many phenomena in which early experience is critical in the shaping of adult behavior.

Patients deprived of the corpus callosum by surgery are unable to describe in speech or writing things that they see in the left half-field of vision. Whereas they have no trouble with items in the right half-field, they consistently report that they see nothing when stimuli are presented on the left side of the vertical meridian. In these tests, the visual stimuli are flashed at 1/10 sec or less to prevent the use of rapid eye movements to get the stimuli into the other half-field. With further testing, however, it becomes evident that these commissurotomy patients are able to speak about their inner experiences from one of their hemispheres only—specifically, the left hemisphere, generally dominant in right-handed persons. Other kinds of tests show that, when the major hemisphere reports that it did not see a left-field stimulus, it speaks for itself alone, and that the stimulus was indeed seen and often well com-

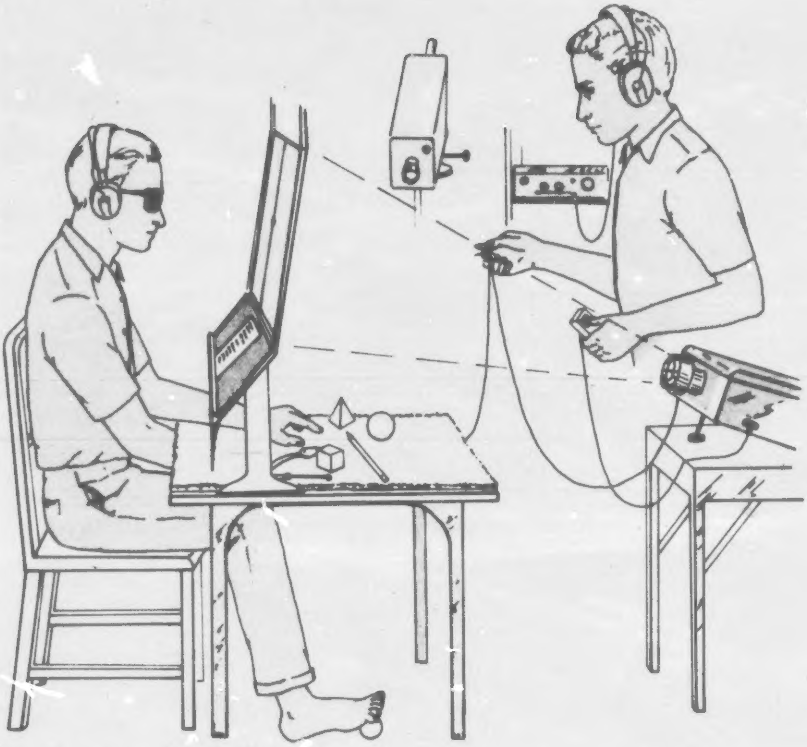
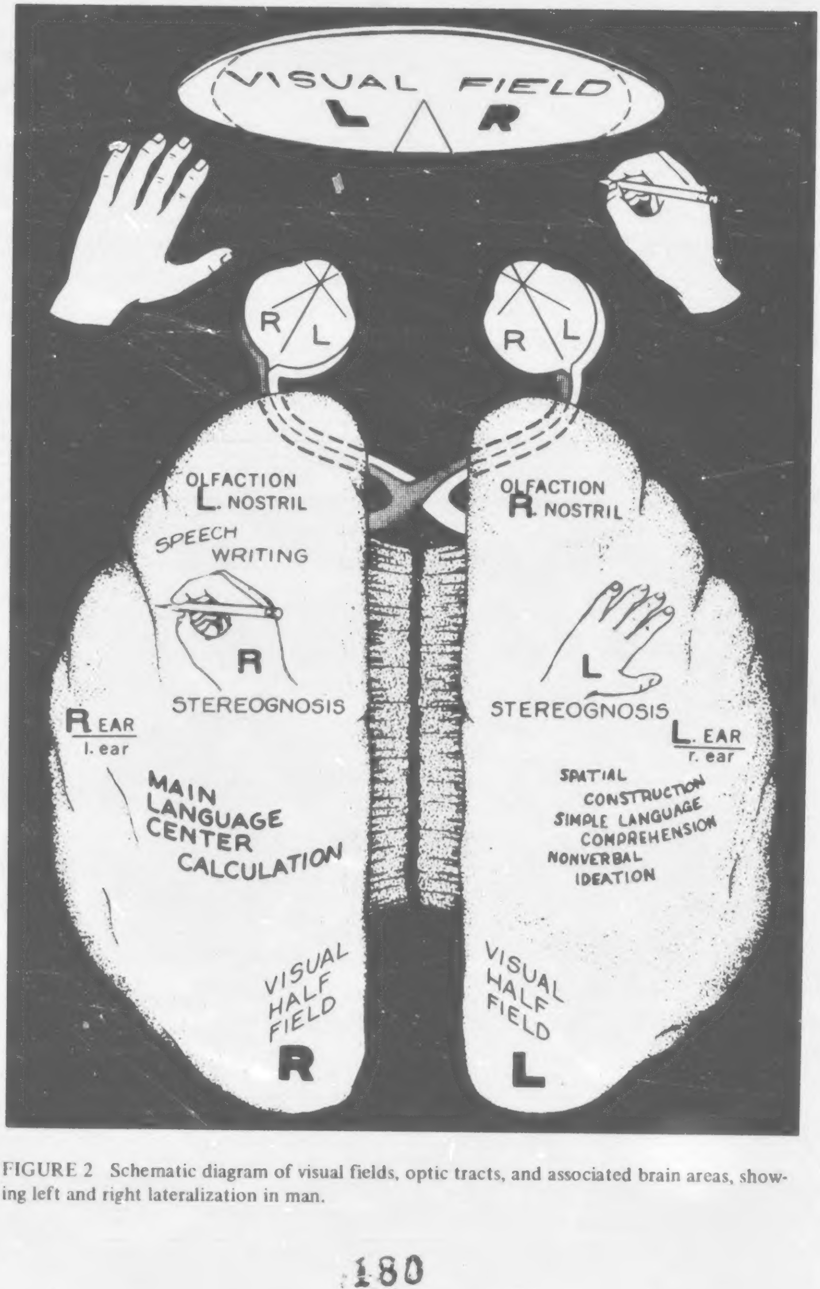


FIGURE 1 Drawing of experimental setup used to demonstrate subject's ability to comprehend a stimulus confined to one visual field.

prehended by the nontalking, the mute, or minor hemisphere. The minor hemisphere's comprehension is expressed in nonverbal tests in which the subject selects the correct name of the stimulus or a matching picture or object by pointing. Ability of the subject to retrieve by touch alone objects pictured in the left half-field and emotional responses to left-field stimuli also show that the left-field stimuli, about which the subject verbally disclaims any knowledge, are actually seen and recognized in the minor hemisphere. Figure 2 shows some of the relationships diagrammatically.

The inner visual world of these subjects has been inferred from such evidence to be double, rather than single, with a separate conscious


VISUAL FIELD
L
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SIMPLE LANGUAGE
COMPREHENSION
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FIGURE 2 Schematic diagram of visual fields, optic tracts, and associated brain areas, showing left and right lateralization in man.

visual awareness in each hemisphere. The right and left inner visual spheres lack their normal conscious connection. A profound absence of awareness in each hemisphere of the mental experiences of the other is consistently evident in the test data. Along with the immediate visual perception, visual memories and all kinds of mental associations of vision with language, with calculation, and with other sensory modes—including touch, hearing, and olfaction—are all confined to the same hemisphere.

No evidence of a similar separation and doubling of inner experience, visual or otherwise, was found in our patient with congenital absence of the callosum. She gave verbal reports from either visual half-field with no hesitation. She was able to read words and numbers across the vertical meridian with no sign that right and left halves were perceived separately. Unlike the surgical patients, she could retrieve with either hand objects seen in either visual half-field. She could add and multiply pairs of numbers shown one in the left and one in the right half-field. In tests involving stereognosis and auditory and olfactory input, she also displayed seemingly normal right–left cross integration.

The extent to which functional compensation has been achieved in this patient's vision is perhaps best illustrated by tests that involved the rapid reading of words presented tachistoscopically, part of the word to the left and part to the right of the vertical midline. For example (see Figure 3), the letters "a b" might fall in the left field for projection to the right hemisphere, and the letters "o v e" in the right field for projection to the left hemisphere. In the same list may be other words like "stout," "only," and "rely," so that the subject cannot use the left or

L YE
AB OVE
ST OUT
ON LY
RE LY
AL IGN
RE IGN
AL LY

FIGURE 3 List of words used to test subject's ability to integrate stimuli coming from left and right visual fields.

right part of the word to cue in the whole word. Even the pronunciation of a syllable or two seen on one side of the vertical meridian often cannot be inferred without consideration of the rest of the word, on the other side. Both parts of the word must thus be taken into account and integrated into a proper whole. S.K. was able to read these words with the mixed right–left input promptly and as well as with the unified right or left input.

We can see the extent to which callosal compensation had been achieved, but we cannot yet explain it satisfactorily. The radical difference in the functional symptoms produced by congenital and by surgical separation of the hemispheres appears to be a direct reflection of the greater plasticity of the developing nervous system, compared with the fully developed system. The underlying factors responsible are very likely basic to many of the more general phenomena that illustrate the functional plasticity of neural maturation. Any insight into the underlying neural factors in this or any other situation would have importance for the whole field of developmental psychobiology, with wide implications extending into ethology, psychiatry, pediatrics, and other disciplines concerned with the effects of early experience on adult behavior.

To account for the compensation achieved in patient S.K., we had best start by reaffirming the absence of any readily apparent explanation. Her normal or near-normal performance on the tests mentioned remains puzzling and difficult to account for in terms of the anatomy and physiology of known neural pathways. Although the anterior commissure often is also absent in such cases, it appears to be present in this person, judging by her x-rays, and to be slightly enlarged, as is not uncommon among cases of agenesis of the corpus callosum. The extra fibers in the anterior commissure might thus be a contributing factor. However, these extra fibers probably total less than 2% of the missing callosal system and have only indirect cross-connections for many of the functions tested. Accordingly, we must look much further for a full explanation. The hippocampal, posterior, and other cerebral commissures apparently are not subject to hypertrophy in callosal agenesis.[3] In an asymptomatic case examined microscopically by Slager *et al.*,[5] the two hemispheres were found to exhibit an essentially typical cytoarchitecture, except for the missing commissure fibers. The histologic examinations described to date, however, have generally been rough, and they do not rule out the presence of an enrichment and elaboration of commissures and decussations at midbrain and lower levels.

To account for the observed degree of functional compensation in S.K., it would seem necessary to postulate at least a functional elaboration of brain-stem and perhaps lower cross-connection systems. The thinness of the cerebral aqueduct and the ease with which it becomes blocked make one wonder about the presence of an atypical hypertrophy among the midbrain centers. In addition to a purely functional reinforcement of whatever connection possibilities exist at these lower levels, there might also be purely embryonic reactions associated with the agenesis of the neocortical system that would make for an enhanced development of the older brain-stem systems that handled higher visual, auditory, somatesthetic, and other functions before the neocortex evolved.

Hypertrophy or functional reinforcement of the normally weak ipsilateral sensory projection system would go far to account for the observed compensation. The behavioral results of early, compared with late, hemispherectomy illustrate the capacity for such development in the somatesthetic system. The ipsilateral auditory and kinesthetic components are already highly developed, and their enrichment would seem to offer no problem. To attain an adequate ipsilateral function in the visual mode would pose the greatest problem, and for present purposes we can focus on the observed visual cross-integration and possible explanatory factors.

The observed ability of S.K. to rapidly read words that fall partly in one half-field and partly in the other seems to imply that the ipsilateral half-field had become projected into the same hemisphere as the opposite half-field. It follows that both half-fields must be closely integrated with speech, also in the same hemisphere. Possible anatomic pathways for this are not easy to see.

One remotely possible pathway for such cross-integration is the anterior commissure. This commissure cross-connects the temporal lobes that are known to be involved in vision. The route is indirect, however, and it is unlikely that the requisite sensory information could be transmitted in sufficient detail to permit one hemisphere to read letters projected from the other hemisphere through the anterior commissure.

Better possibilities probably can be found in cross-connections at midbrain levels associated with visual function in the superior collicular, pretectal, and pulvinar systems.[9] Before evolutionary development of the neocortex, the midbrain systems carried out visual integration at the highest levels. The upper levels of midbrain vision in present mammals are difficult to assess because of close interaction with the neocortex and dependence on cortical connections. In any case, it is important that visual deficits produced by neonatal removal of occipital lobes are much smaller than those produced by adult removal.[10] We are speaking here mainly of the focal identifying type of vision, rather than the orientational sort more characteristic of the midbrain in the cortically intact mammal.

Assuming that a latent potential for high-level focal vision in the midbrain may be evoked by agenesis of the callosum, as well as by early cortical damage, there would still remain the problem of getting the refined pattern information for reading small letters across the midline, up to the cortex, and integrated with the contralateral information for a verbal readout.

In our latest tests for visual cross-integration, I have used only two- and three-letter words, in an effort to avoid the variables introduced by peripheral vision. The initial scores of S.K. under these conditions show an encouraging difference between the lateral unified input and the mixed or combined right–left input, indicating that in these near-threshold performances that might separate midbrain from direct cortical channels she handles the left-field input better than either the right-field or the combined right–left input.

The question arises of whether speech is bilateralized in this patient. Conclusive evidence is lacking. She is ambidextrous to a high degree, as is often the case in patients with agenesis of the callosum. For example, she writes mainly with her left hand, but she uses scissors better with her right hand. Some preliminary evoked-potential records taken during visual performance suggest that only her left hemisphere is active in vision. It is conceivable that speech, somatesthesis, audition, and vision are all handled in a single dominant left hemisphere. This fits with the findings on near-threshold reading of words from combined right- and left-field input.

Regardless of whether speech is bilateralized or is developed only in a dominant hemisphere, there are indications that in S.K. speech has been developed at the expense of other mental faculties, such as spatial perception. After we had established the lack of functional deficits in the regular series of tests used to demonstrate cross-integrational symptoms after commissurotomy, we started to administer other types of tests—more generalized tests aimed at the upper limits of various mental and sensorimotor faculties, regardless of lateralization, following the approach of Jeeves.[1] The results to date are only suggestive, but they begin to point to subnormal function in a number of nonverbal capacities. S.K. fairly consistently does better on verbal tasks than on performance or perceptual tasks. She also draws poorly and has difficulty with geography, block design arrangements, and matching patterns—all specialties of the minor hemisphere in typical right-handed persons.

At this stage, our evidence suggests a distinction between two somewhat different types of cross-integrational functions mediated by the corpus callosum: those which can be compensated for in congenital absence of the corpus callosum and those for which compensation is more difficult or impossible. The kinds of functions for which compensation is achieved involve the more direct sensory and motor cross-integrations that were carried out at subcortical levels before evolution of the neocortex. When the neocortical system for vision, normal stereognosis, and other functions evolved, their cross-integrational mechanisms also had to be moved upstairs. The kinds of cross-integrational functions for which compensation is not so easily achieved are those associated with cerebral dominance and the lateral differentiation of higher mental faculties that is peculiar to the human brain. Particularly affected are performances that depend on the mental faculties specialized in the minor hemisphere.

If S.K. has double speech—that is, bilateralized development of speech in both hemispheres—or if speech in her dominant hemisphere has no direct cross-communication with the other hemisphere, the results are much the same. In either case, there is a handicap in that the verbal activities cannot be so well reinforced by functions for which the minor hemisphere is normally specialized, owing to lack of cross-talk in the former case and to intrahemispheric competition in the latter. These functions of the minor hemisphere seem to include spatial and orientational activities, abstract thinking, and creative mathematical and geometric abilities, all of which normally would cooperate with and embellish the verbal hemisphere through the corpus callosum. It is pertinent that, according to the literature, even the least symptomatic subjects with agenesis of the corpus callosum have not been brilliant or even above normal in intellect. The current view is that they attain mediocre intelligence at the most, although they may be highly verbal and even multilingual.

The interpretation that loss of the corpus callosum prevents reinforcement by minor hemisphere functions fits also with results of some recent work.[2] We have considerable evidence that the functions of the minor hemisphere are sufficiently different in kind from those of the major hemisphere that the two tend to conflict and interfere with each other, making it a real advantage to put the two types of activity in separate hemispheres. The minor hemisphere seems to be a specialist at configurational, spatial, synthetic, and geometric activity, whereas the major hemisphere is specialized for sequential, verbal, logical, and analytic activity. The two functions do more than compete for brain space in evolution; the basic difference in the nature of their organization means that excellence in one tends to interfere with top-level performance in the other. On the basis of evidence collected from patients with congenital and surgical absence of the corpus callosum, as well as from the literature, this fundamental antagonism in the nature of these modes of brain functions might be a causal factor behind the evolution of cerebral dominance and lateral specialization in the human brain.

What meaning this may have for problems of dyslexia remains to be seen. One wonders whether a possible factor in dyslexia is an overly strong or extensive, perhaps bilateral, development of the verbal, major-hemisphere type of organization that tends to interfere with an adequate development of spatial gnosis in the minor hemisphere. The facts that general verbal capacity tends to be good in dyslexics and that the frequency of dyslexia is higher among left-handed persons would fit such an interpretation. Extra training in spatial gnosis with special reference to alphabet patterns and the troublesome letters and words subject to directional reversals would seem a natural approach to these problems.

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DISCUSSION

DR. INGRAM: I suggest that the dyslexic individual is faced with several problems in addition to the spatial relationships that he cannot visualize. He also requires some teaching in terms of auditory concepts, because this function is in the hemisphere that is functioning best and that should be concentrated on.

DR. MASLAND: Dr. Ingram has suggested that there is a very important additional element in the problem of reading. It is not merely a matter of recognizing the shape of an object, but the establishment of an association between the shape of an object and a verbal sound information element. That function has already been established in the left hemisphere of the average child, and it seems to me that the fundamental problem of the dyslexic child, particularly the child who is having letter reversals, is the necessity of establishing a relationship between a visual spatial function, which is most likely mediated in the right hemisphere, and a language function, which has already been established in the left hemisphere. The problem is to develop techniques whereby spatial functions are dissociated from the language functions of the left side.

DR. INGRAM: There is a possibility that this dyslexic schoolchild would be taught by the so-called "look and say" method. I think that would be disastrous. This child has to be taught to relate the visual symbol, as Dr. Masland says, to the spoken syllable. I think by the stage of learning to read the child is probably able to recognize the visual symbol, but not to relate it to the auditory symbol. Therefore, you have to work with a phonic approach, and establish the phonic relationships. I am trying to point out that it is difficult to short-circuit a function that you think is not there.

DR. SPERRY: I was referring specifically to the perception of spatial relations during early learning of reading and writing, when letters and words tend to be reversed, and did not mean to imply a general application to reading aloud and to all forms of dyslexia.

DR. MASLAND: Maybe it is unwise to generalize, but for a person to learn to read, obviously he has to have the ability to see, he has to be able to analyze and recognize the material being seen, and he must be able to associate that object with an auditory symbol.

DR. BERING: The auditory counterpart is not necessary. It has been brought up here that people can learn to read without it if the hemispheres are intact. There are auditory and spoken relationships with reading, but neither is absolutely necessary.

ATTENTIONAL AND PERCEPTUAL MECHANISMS

RICHARD JUNG / LOTHAR SPILLMANN

Receptive-Field Estimation and Perceptual Integration in Human Vision

In the visual system, the sensory coding of luminance differences is based on the organization of receptive fields in two neuronal subsystems, "B" and "D," sending reciprocal information from the retina to the brain. The B system, consisting of on-center neurons, signals "brighter," and the D system, consisting of off-center neurons, signals "darker."[2,18] The coding of contrast is accomplished by lateral inhibition and activation: B neurons are inhibited by illumination of their receptive-field surround, and D neurons are activated.

The receptive fields of visual neurons in animals and man have been investigated mainly by three research methods: the extent and organization of the retinal areas projecting to individual neurons were determined, the transformation of the receptive-field organization was studied at different levels of the central visual system, and indirect estimates of the size of receptive fields in man were obtained and correlated with results from animal experiments.

The first method was inaugurated by Hartline,[11,12] who defined a receptive field in the frog as that area on the retina within which illumination activated or inhibited an optic-nerve fiber. This concept was particularized by the description of lateral inhibition in *Limulus.* It was further refined in experiments by Kuffler[22] and others on the antagonistic organization of field center and surround in cats and monkeys. The second method was introduced in 1959 by Hubel, Wiesel, and Baumgartner, who studied the neurons of the retina, the lateral geniculate nucleus, the primary visual cortex,[15,16] and the paravisual cortex[17] (areas 18 and 19) in the cat. Similar neuronal recordings from cortical cells in man by Marg *et al.*[23] are discussed elsewhere in the proceedings. The third method was developed by Baumgartner,[1] who, from his animal experiments, derived indirect procedures for investigating human receptive-field organization. The following report is concerned mainly with this third line of research in man; however, for a better explanation of the basic neuronal mechanisms, it will also include some related results obtained in animals. In human vision, we are virtually restricted to the psychophysical approach.

RECEPTIVE-FIELD ESTIMATION IN MAN BY HERMANN'S GRID

A simple method of determining the size of visual receptive fields in man is by means of contrast patterns viewed from different distances or under different angles. Baumgartner,[1] using the Hermann grid,[14] was the first to measure foveal field centers in this manner (Figure 1). Several workers from our department—Kornhuber and Spillmann,[21,28] Sindermann and Pieper,[27] and others—have since estimated the size of field centers, as well as surrounds, by this or related indirect methods. With the grid technique, receptive-field centers in the fovea were found to be 25-30 μ in diameter, and centers plus surrounds, about 50 μ. These values correspond to 5–10 min of arc of angular projection.[1,27,28] Receptive-field centers in the extrafoveal regions of the eye appear to be much larger. Mean diameters increase linearly toward the periphery, doubling their size from 1.5 to 3 deg between 20 and 60 deg of retinal eccentricity (Figure 2).

Bryngdahl's data[5] referring to sine-wave contrast patterns yield diameters of 20–100 μ for foveal and parafoveal fields and agree with results obtained by Baumgartner[1] and Sindermann and Pieper[27] with different methods. Direct measurements of receptive fields of a small number of human cortical cells were reported by Marg *et al.*[23] They were in extrafoveal regions and appeared to have ill-defined borders.

Hermann grid stimulation of concentric-field neurons in the lateral geniculate nucleus and primary visual cortex of the cat shows strongly

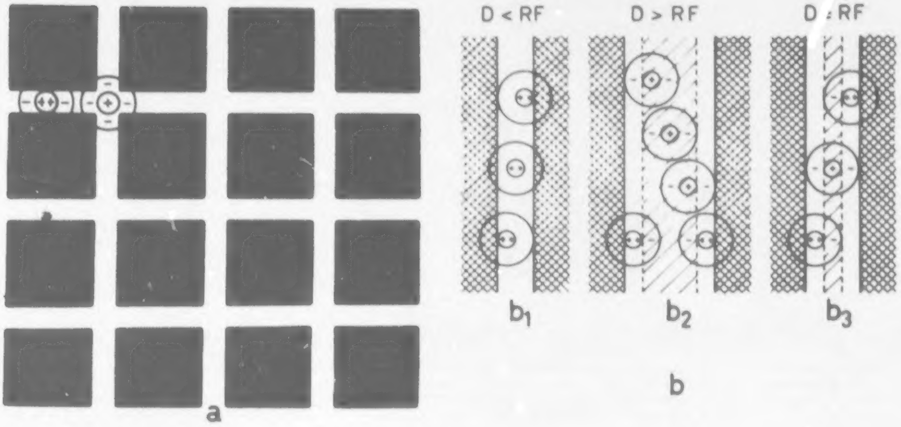

FIGURE 1 Estimation of receptive fields and field centers in man by simultaneous brightness contrast. The Hermann grid (a) and the array of white stripes flanked by dark surrounds (b) are modified drawings of experimental patterns used in studies by Baumgartner[1] and Sindermann and Pieper.[27] (a) In the Hermann grid, gray spots are seen at the intersections of the white bars, except when fixated foveally. This peculiarity can be explained by receptive-field centers of different size. Projections of receptive fields are shown in two critical positions. They illustrate schematically how differences in lateral inhibition may account for the darkening by producing twice as much neuronal activation (++) on bars, compared with intersections (+). In the fovea, receptive fields are much smaller than in the extrafoveal retina. Consequently, their inhibitory surrounds have similar effects, whether illuminated by bars or intersections (see example in b_2). (b) The projection of a foveal receptive field is shown in different positions relative to a white bar of various width. (b_1) Maximal brightness (++) due to a minimum of lateral inhibition occurs when the angular size of the white stripe approximates or matches the diameter of the receptive-field center. (b_2) In wider stripes, a gray central canal ("inner contrast") is seen accompanied by enhanced contours on either side ("border contrast"). It indicates local differences in total neuronal activation resulting from complete or partial lateral inhibition. (b_3) When the diameter of the receptive field equals the angular width of the white stripe, inner contrast is most distinct. (Sometimes it can be also observed in the Hermann grid, in addition to the darkening at the intersection.) To see the various contrast phenomena, one should look at Figure 1a from distances ranging from 10 to 60 cm.

reduced response rates at intersections and relative enhancement for either of the two bars (Figure 3). These neurophysiologic results compare favorably with apparent brightness differences in human vision.[19] In contrast, a subjective correlate of receptive fields with oblong forms and elongated axes, described by Hubel and Wiesel[15] in area 17 of the cat, cannot be ascertained in man by the grid technique.[19] In cats, grid stimulation of these "simple-field" neurons produces neuronal enhancement only in bars that in position and orientation coincide with their

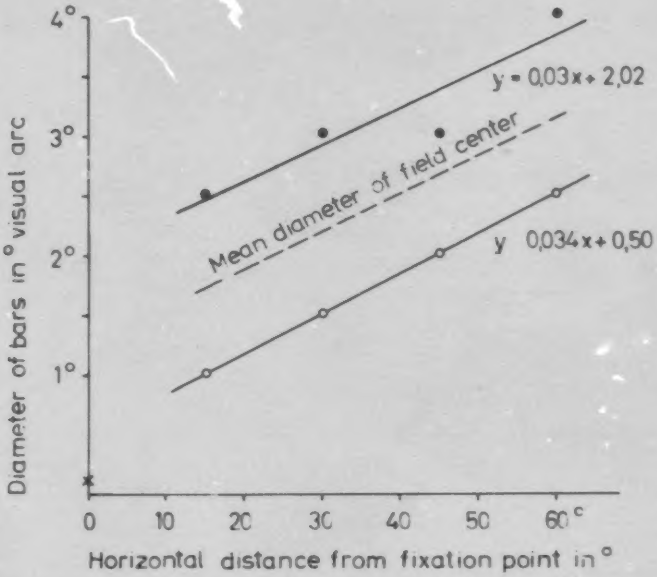

FIGURE 2 Lower and upper thresholds for the Hermann grid illusion as a function of retinal eccentricity (from Spillmann[28]). The Hermann grids in this experiment (other than in Figures 1 and 3) consisted of black bars presented against a white background. Grids of various stripe width were shown at different horizontal distances from the fixation point. Observations by one subject were made with artificial miosis to compensate for hyperopia in the peripheral retina. The critical bar width at which the illusion appears (circles) or disappears (dots) increases almost linearly with eccentricity.

receptive-field axes. Also, less response diminution is found at grid intersections, except in large receptive fields.[26]

RECEPTIVE-FIELD ESTIMATION BY APPARENT MOTION

Wertheimer's apparent motion[29] elicited by two successive light stimuli presented at different loci in the visual field was used to estimate the size of receptive fields for movement perception. The maximal distances between the alternating spots across which object motion ("optimal" or "beta" motion) or pure motion ("phi phenomenon") could be seen were determined as a function of retinal eccentricity. Figure 4 shows that threshold distances for both types of motion increase linearly toward the periphery, doubling their size between 20 and 60 deg of eccentricity. Values for phi are somewhat larger than for beta motion.

The "receptive fields" for Wertheimer's apparent motion increase toward the periphery of the eye at the same rate as the receptive-field centers determined by the Hermann grid. Both measures show an increase by a factor of two between 20 and 60 deg of retinal eccentricity. In absolute terms, the field sizes for apparent motion are approximately 10–20 times larger than those for simultaneous contrast (Figure 5).

The physiologic basis of these large fields seems to be a temporospa-

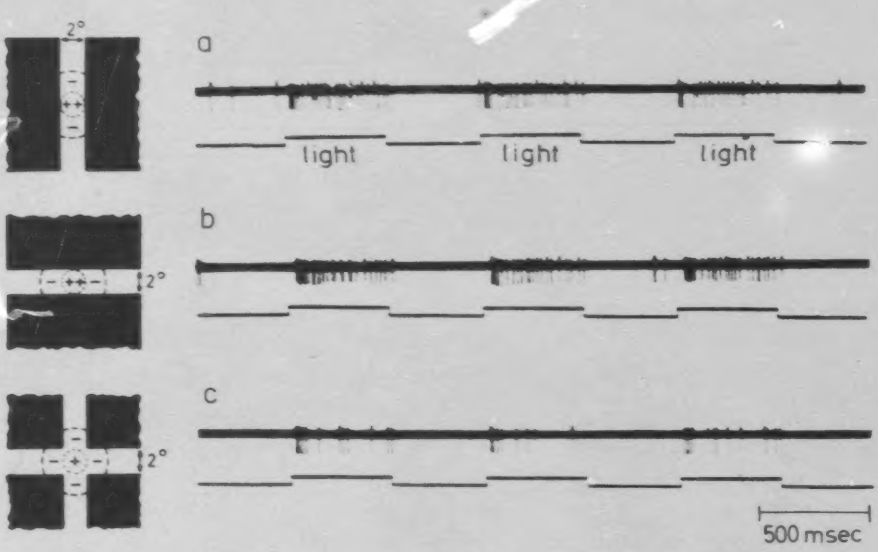

FIGURE 3 Response of a first-order B neuron in the visual cortex of the cat to various positions of the Hermann grid within its receptive field (from unpublished experiments by Baumgartner). The discharge rate of this neuron is consistent with the subjective brightness diminution seen at the grid intersection. (The receptive field in this example had a diameter of 6 deg and was located 20 deg paracentrally.) In positions a and b (bars), the response to light is more than twice as strong as in position c (intersection). These results are accounted for by differences in surround illumination and lateral inhibition. The behavior of this neuron is typical only for concentric fields of geniculate and first-order cortical B neurons. In the oblong simple-field neurons of Hubel and Wiesel,[15] the response depends on stimulus orientation. It reaches a maximum when the white bar coincides with the receptive-field axis. The response is minimal when bar and field axis are oriented at right angles to each other and is intermediate when stimuli a and b are combined in a pattern of two intersecting bars.

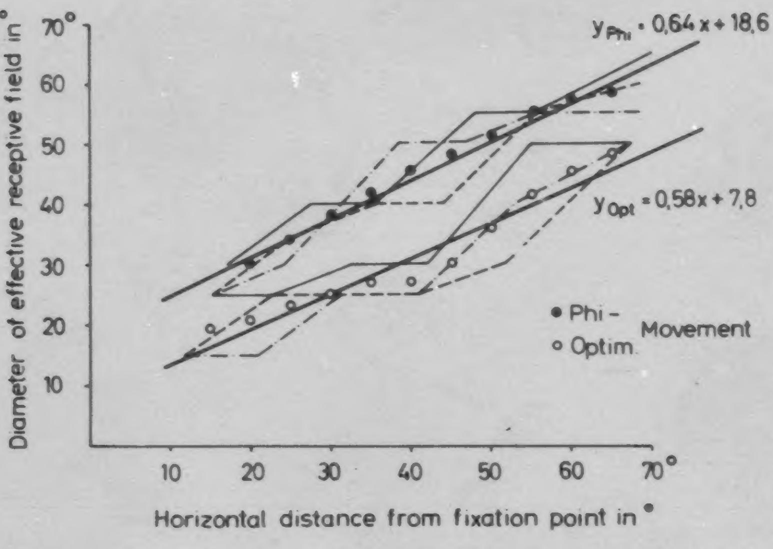

FIGURE 4 Maximal angular distances for apparent motion as a function of retinal eccentricity (adapted from Spillmann[28]). Upper thresholds for apparent motion were determined with two alternating lights 3 deg 20 min in diameter presented with an interval of 240 msec. Fixation was on a vertical line between the two stimuli. Criteria were the perception of object motion (optimal, or beta) or pure motion (phi phenomenon). Mean thresholds of three subjects indicate that the critical distances across which motion is seen are somewhat greater for phi (dots) than for beta (circles). Both types of thresholds show a nearly linear increase in size with retinal eccentricity.

tial network of many interacting neurons arranged to signal the successive occurrence of photic stimuli as motion of particular direction and velocity. These neuronal populations may normally require physical movement for adequate stimulation, but under some conditions respond also to a sequence of two light spots. The extent of neuronal convergence causing these motion-sensitive neurons to function as a unit or receptive field can be estimated only with reference to the special dimensions used (spot diameter, 3 deg 20 min; sequential interval, 240 msec) and may vary for other conditions. In our experiment, the linear velocity corresponding to the critical spot sequence for maximal separation was a function of retinal eccentricity and ranged from 60 to 240 deg/sec. This is in the upper range of and even beyond the highest human velocity estimates investigated psychophysically by Dichgans *et al.*[7]

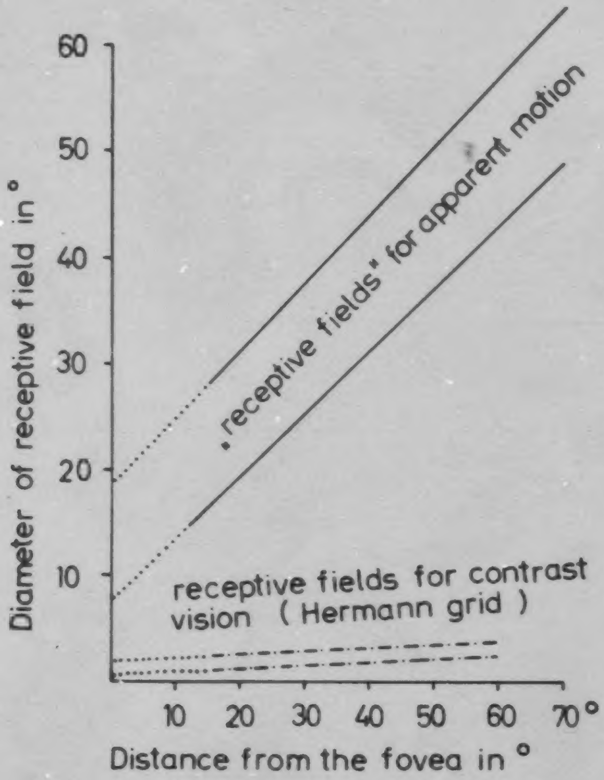

FIGURE 5 Comparison of human receptive fields and field centers for contrast vision (Hermann grid illusion), apparent motion (beta movement, phi phenomenon), and eye pursuit movement as a function of retinal eccentricity (adapted from Spillmann[28]). Mean values for the first and second procedure were derived from data shown in Figures 2 and 4. Eye pursuit movement (dashed line) was elicited by a vertical-line stimulus oscillating on a horizontal meridian with a sine-wave frequency of 0.3 sec. Results obtained with this third method represent minimal amplitudes of the stimulus required for a correlated pursuit movement. In spite of differences in absolute size, thresholds for all three procedures show an approximate increase by a factor of two between 20 and 60 deg of retinal eccentricity. This figure compares favorably with direct measurements of receptive-field centers in monkeys (Hubel and Wiesel[15]), suggesting that both objective determinations and indirect psychophysical estimates may refer to the same basic neuronal organization.

EHRENSTEIN'S BRIGHTNESS ILLUSION IN THE ABSENCE OF PLANE CONTRAST

Ehrenstein[8] in 1942 experimented with patterns of radial lines that cause brightness enhancement at the white center spot to which they converge (Figures 6 and 7). The lines must exceed a particular length (Figure 6, left), and there should not be fewer than four. Usually, the central spot is seen as a round patch within which the brightness enhancement occurs. This round blob is altered to an apparent square when the lines are thickened and physical contrast between adjacent areas of black and white becomes more intense (Figure 7). Paradoxi-

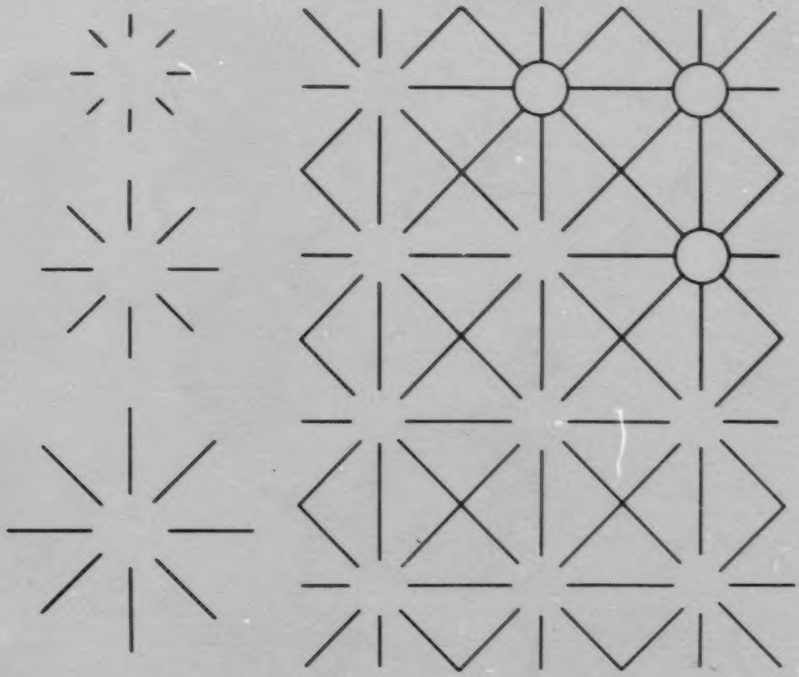

FIGURE 6 Ehrenstein's brightness illusion[8] in the center of radially converging black lines. Central spots appear brighter than adjacent white areas if one views the pattern freely. As in the Hermann grid, the illusion becomes less apparent when the central area is fixated. Paradoxically, the brightness enhancement disappears when the center spot is surrounded by a thin black circle (upper right). A minimal length of lines is required to induce the illusion (left).

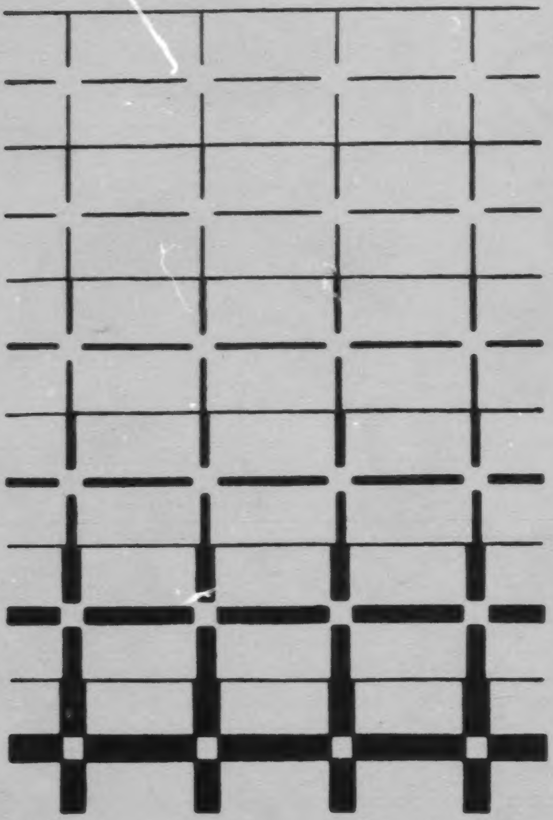

FIGURE 7 Ehrenstein's illusion as a function of line width – a brightness paradox. Physical contrast between figure and ground is strongest in the lower part and weakest in the upper part. In spite of this brightness, enhancement of the central spot appears to be most vivid in rows 3, 4, and 5, followed by the rows at the top and then at the bottom. There are inter-individual differences in sequence. At close fixation, brightness enhancement is replaced by another illusion forming a gray diagonal cross (X) within the central area.

cally, under these conditions the central spot is less enhanced. It becomes even more inconspicuous when it is completely enclosed by black bars (Figure 7, bottom). Brightness enhancement disappears entirely when the central area is surrounded by a thin circle (Figure 6, upper right).

Sometimes a quite different illusion appears in Figure 7, most readily at the third, fourth, and fifth rows and with central fixation. Instead of the brightness enhancement, a grayish cross (×) emerges, connecting the edges of the apparent square along the diagonals. It is best seen from about 30 cm or farther away. At shorter distances, it disappears and thus may depend on the angular size of the foveal projection. This cross illusion was not described by Ehrenstein.

It remains for further experimentation to decide whether the phenomenon may be interpreted in terms of interactions between line stimuli, described as neural interaction in the human fovea by Fiorentini and Mazzantini.[9] It is of interest that the cross illusion appears to be confined to the fovea. In contrast, the brightness illusion fades during prolonged foveal fixation, requiring eye movements for revival.

DISCUSSION

The Hermann Grid Phenomenon: Single-Cell Explanation versus Population Hypothesis

The Hermann grid illusion is an example of brightness contrast attributable to large groups of visual neurons. For a single nerve cell, the phenomenon can be explained by lateral interaction within its receptive field. For neuronal populations, however, the explanation is more complex and must concern both neuronal subsystems, the one signaling brightness (B) and the one signaling darkness (D). The Hermann pattern not only elicits a diminished brightness sensation at the intersection of white bars; after figure–ground reversal, it also results in a diminished darkness sensation at the same (now black) intersection. Neurophysiologically, the illusion is based on different distributions of lateral inhibition or activation in the surrounds of on-center fields (B neurons) and off-center fields (D neurons), respectively. Retinally and postretinally, these two subsystems interact in a manner yet unknown.

Purely psychophysical methods in human vision will hardly reveal at which level within the visual system this interaction takes place. However, neuronal recordings in the cat suggest that the grid phenomenon relates to a concentric receptive-field organization mainly in the "lower" parts of the visual system (retina, lateral geniculate nucleus, area 17). The more elaborate neuronal systems of the visual cortex showing transformations from concentric to oblong and complex field organizations appear to be less involved in the Hermann grid illusion. Only the simple fields of Hubel and Wiesel ("line detectors") may still retain some error of brightness information,[26] thus indicating that the neuronal coding of luminance differences is not excluded from the cortex.

Other conditions, mainly afterimages and eye movements, may have an influence on the Hermann grid phenomenon. These factors are investigated by Sindermann in a forthcoming publication and will not be discussed here. Neuronal integration of receptive fields from concentric to simple and complex field organizations requires transmission through several synapses. The role of inhibition for this information processing is not confined to lateral inhibition from the field surround. Other types of inhibitory action have been demonstrated by electric stimulation of the optic radiation. For simple-field neurons, response latency is at least one synaptic delay longer than for concentric-field neurons.[6] Complex-field neurons usually show long primary inhibition after shock stimulation of radiation fibers.

To avoid confusion between neuronal receptive fields and their possible equivalents in human perception, a terminologic distinction might be useful. We tentatively propose the term "perceptive fields" for the subjective correlates of receptive fields estimated in human vision.

The Ehrenstein Illusion and Its Dependence on Oriented Lines

Since 1870, the Hermann grid illusion has been explained by simultaneous contrast of bright and dark areas that, according to Baumgartner, stimulate concentric receptive fields of visual neurons. Ehrenstein's brightness illusions, which appear within oriented line patterns in the absence of contrasting planes, cast considerable doubt on this explanation by contrast alone.[8] Brightness enhancement in the center of radially converging lines can hardly be explained on the basis of circular receptive fields. Detrimental effects of steady fixation and enclosing circles on the illusion are rather reminiscent of Hubel and Wiesel's findings[15,16] of simple-field neurons that respond optimally to lines of given orientation moving across their receptive-field axis.

In the absence of a sufficient neuronal explanation of the Ehrenstein illusion, one may speculate that these neurons do not only signal edges, lines, and orientations, but also may contribute to brightness sensation. A neurophysiologic basis might be that the ellipsoid fields of these neurons can be divided into subclasses carrying brightness and darkness information, respectively: B neurons with oblong on centers and D neurons with oblong off centers,[20] both transformed from corresponding subsystems of the retina and lateral geniculate nucleus.

Receptive Fields and Levels of Neuronal Interaction within the Visual System

The rather simple concept of the receptive-field organization of a single neuron might be considered a model for similar interactions involving collateral inhibition and activation among neuronal populations. It seems worth noting that lateral inhibition and activation as the essential mechanisms of the receptive-field organization in retinal ganglion cells can still be demonstrated in the visible phenomena of contrast and contour vision. Hartline,[13] in his first short note in 1949, had already drawn attention to the role of lateral inhibition in contrast vision. Since then, many studies of receptive-field organization have confirmed this mechanism and stressed its significance in various species, but without elucidating the central process. How the information of primary lateral inhibition is used in the central nervous system and its large populations of visual neurons is as unclear as the synaptic mechanisms of cascade-like transformations of receptive fields. Only a few facts are known about single-neuron interaction with lateral, collateral, and reciprocal inhibition in the two neuronal systems, B and D. It is unlikely that any of them singly or in combination can sufficiently explain the integrated effects in thousands and millions of neurons necessary for sensation and perception.

Results of recent experiments on Mach bands with flicker photometry favor the localization of lateral inhibition and mutual interaction of receptive fields beyond the receptor organ. After measuring the brightness of Mach bands, von Békésy[4] proposed the existence of lateral interaction in brain structures. The rather complex transformation of receptive fields at various cerebral levels and the role of eye movements and of inhibition within the field center cannot be discussed here. We mention only two findings: Richards[25] has described modifications of area summation in man during accommodation and convergence that he explains by plasticity of receptive fields. Freund and co-workers[10] demonstrated a special transformation of field organization in the cat's D neurons of the lateral geniculate nucleus that differ from retinal off-center neurons by showing marked off inhibition, instead of spatial summation.

Generally, lateral inhibition in the retina and collateral inhibition in cerebral structures may, in spite of their differences as neurophysiologic mechanisms, have similar effects on visual contrast and perceptual integration. In principle, this similarity holds even for different sensory modalities, as demonstrated by von Békésy[3] in his work on sensory inhibition.

RECEPTIVE FIELDS IN RELATION TO FORM VISION AND VISUAL LEARNING

The neuronal mechanism of visual learning is unknown. Thus, it seems difficult to discuss its interaction with neuronal processes of sensation and perception. However, the importance of visual memory for form vision, perception, and reading is obvious, and relationships to receptive-field transformation are conceivable. A child learns to read by organizing and recognizing patterns composed of lines in different orientations. Perceptually, this task involves both "innate" neuronal mechanisms and new connections acquired during the learning process. Wiesel and Hubel's experiment[30] showed that cats have innate "simple" receptive fields responding optimally to line stimuli of particular axis orientation. These neuronal line detectors are present in newly born kittens, before any visual experience, but deteriorate when contrast patterns are excluded from vision during the first months of life.[30] Thus, visual learning apparently maintains and facilitates visual function in the cortex during the early periods of life. Short- and long-term visual memory not only are necessary for acquiring form recognition, but also are prerequisites for the normal function and early development of innate neuronal coordination. In this context, it may be of interest to ask whether the above-mentioned Ehrenstein illusions, if dependent on line detectors, are influenced by visual learning.

In discussing learning mechanisms in relation to the visual receptive-field organization, some recent experiments in humans on the effect of learning and expectation on Wertheimer's apparent motion should be mentioned. Besides the fact that expectation and bias affect the occurrence of apparent visual motion, Raskin[24] demonstrated long-term memory effects on special patterns seen previously. Prior experience as old as 1 week either facilitated or interfered with subsequent perception of apparent motion. Raskin concluded that the perceptual feature of motion or nonmotion may become associated with certain form characteristics by way of learning. We cannot discuss here the rather complex neurophysiology of motion perception and its dual mechanisms–afferent movement caused by passive displacement of retinal images and efferent movement produced by active pursuit movements of eye, head, and body. Both mechanisms appear to be intimately linked to the detection of contrast, allowing one to fixate stationary and moving borders. Thus, it is not surprising that the minimal threshold amplitudes for eliciting eye pursuit movement[28] are of the same order of magnitude as the receptive-field diameters obtained with the Hermann grid illusion (Figure 5).

Although learning should influence form recognition and might be involved in the progressive transformation of receptive fields, we cannot yet apply our results to the physiology of reading and its disorders. Because we read with moving eyes during short pauses of fixation, making use of black–white contrast for the recognition of letters, we may say only that oculomotor functions and mechanisms of contrast and pattern vision, among others, contribute to the physiology of reading. Whether in man, as in the cat, the organization of simple receptive fields with specific axis orientation is a congenital neuronal property of the visual cortex, requiring early visual experience to prevent deterioration, cannot be answered with certainty. Assuming that a combination of inherent mechanisms with learning is necessary for the normal functioning of the visual system, it might be justifiable to discuss receptive-field organization and its application to human vision in a conference on reading functions.

SUMMARY

Receptive fields of visual neurons as determined by direct recordings in animals can be inferred in man from contrast and movement illusions. Estimates of their spatial extent were derived from threshold measurements for simultaneous contrast and apparent motion.

Diameters of receptive-field centers in the human fovea, when measured with Hermann grids of different bar width, range from 25 μ to 30 μ (5–10 min of arc), and receptive-field centers plus surrounds, about 50 μ.

The size of receptive-field centers is a linear function of retinal eccentricity. Between 20 and 60 deg from the fovea, the average diameter of field centers doubles from 1.5 to 3 deg of arc.

Receptive-Field Estimation and Perceptual Integration in Human Vision

Results of Hermann grid stimulation of concentric-field neurons in the visual system of the cat are consistent with apparent brightness differences in human vision. For white grids, B neurons of the lateral geniculate nucleus and the first stage of the visual cortex show enhanced responses when exposed to bars and diminished responses when stimulated by intersections. Simple-field neurons of the visual cortex show similar enhancement only if the orientation of their receptive-field axes corresponds to border positions of the grid.

Ehrenstein's illusions of brightness enhancement elicited by radial line patterns in the absence of marked physical contrast between figure and ground are tentatively ascribed to cortical simple-field neurons and their possible contribution to brightness perception.

"Receptive fields" determined with Wertheimer's optimal (or beta) motion and phi phenomenon are 20–30 times larger than receptive-field centers measured with the Hermann grid. Analogous to these, "receptive fields" for apparent motion increase linearly toward the peripheral retina, doubling in size between 20 and 60 deg of eccentricity.

The receptive-field organization, by transforming luminance gradients into contrast borders, is a basic mechanism of form vision. Although its significance is evident, its detailed role in pattern vision and reading cannot yet be explained in neuronal terms. Modifiability of receptive fields and plasticity of spatial mapping in the central visual system must be postulated to explain size constancy and form recognition. The possible interaction between memory and neuronal convergence within the visual system is discussed for the example of apparent motion.

The term "perceptive fields" is proposed for the subjective correlates of receptive fields estimated in human vision.

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GEORGE SPERLING

Short-Term Memory, Long-Term Memory, and Scanning in the Processing of Visual Information

A MODEL OF VISUAL-INFORMATION PROCESSING

In reading, as in most visual tasks, the eye gathers information only during the pauses between its quick saccadic movements. The normal input to the visual system is thus a sequence of brief exposures. I would like to propose here a model of the way people process the information they receive in one such exposure. I shall be concerned with the simple situation in which a person is shown briefly an array of letters and then asked to write them and the closely related situation in which he hears spoken letters and is required to write them.

The model shown in Figure 1 summarizes the results of numerous experiments. The squares indicate short-term memories. The first box represents a very-short-term visual memory, which, in the past, I have called visual-information storage.[15] It contains a great deal more information than the subject ultimately will be able to report, but its contents normally fade rapidly, usually within about one fourth of a second. These conclusions are derived from a partial-report procedure: the subject is required to report only a small fraction of the stimulus contents on any trial and does not know in advance which aspects he will be required to report. The methods and results have been described in detail elsewhere.[2,15] It is easily proved that a great deal of information from a visual stimulus gets into the subject's very-short-term visual memory; the information is lost to recall because later processes are unable to use it.

Ultimately, stimulus letters are "recognized"; that is, the subject says or writes them. He makes an appropriate motor response. In terms of the model, it is useful to distinguish between actually executing the motor response (saying, subvocally rehearsing, or writing a letter) and having decided which response is to be executed. This kind of distinction is most often made in discussing computers, and perhaps the terminology that has been developed to deal with it in that domain will help to clarify it here.

Saying a letter may be conceived of as executing a long program that consists of hundreds of instructions to various muscle groups. Recognizing a letter may be considered as having decided which program to execute. In practice, a program is designated by its location, or address: the address is the location of the first program instruction to be executed. The second short-term memory box in the model designates the recognition buffer-memory. It is a short-term memory for letters that are about to be spoken or rehearsed subvocally, i.e., a memory of the addresses of the programs for saying them.

The kinds of data that require the concept of a recognition buffer-

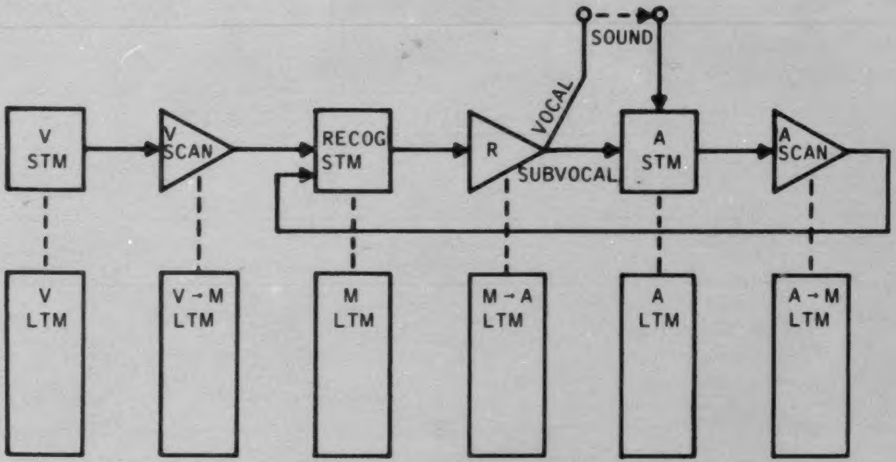

FIGURE 1 Model of visual information processing. Squares indicate short-term memories, rectangles indicate long-term memories, and triangles indicate scan components that transform signals from one modality into another. V, visual; A, auditory; M, motor; R, rehearsal; RECOG, recognition buffer-memory; →, direction of association.

memory have been described.[14] The basic idea is that three or four letters can be recalled from visual presentations even if the effective duration of the presentation—e.g., VIS—is so short that there is not time for the rehearsal of even one letter. The recognition buffer-memory can hold at least three letters (i.e., the addresses of the motor programs for rehearsing the letters) for a period of about 1 sec, until they have been rehearsed.

A scan component is needed to transform the visual information in very-short-term visual memory into the motor-address information of the recognition buffer-memory. The visual scan component is designated by a triangle in Figure 1 to indicate that it is not a memory and that it transforms information from one modality into another. Actually, the visual scan component has at least three distinguishable functions: deciding which areas of the visual field contain information on which further processing should be performed ("prescan"[8]); directing processing capacity to the locations selected by the prescan ("attention"); and converting the visual input from the selected locations into the addresses of motor programs ("scanning").

The maximal rate at which letters are scanned can be measured from visual presentations in which the persistence of the information from an initial letter stimulus is obliterated by a subsequent visual "noise" stimulus. The measured rates are quite high—say, one letter every 10–15 msec, which is equivalent to rates of up to 100 unrelated letters per second [10]

The middle triangle in Figure 1 designates rehearsal. In vocal rehearsal, the motor instructions designated by the recognition buffer-memory are executed, and a spoken letter results. Because it indicates a change of modality or dimension, a triangle is used to designate the rehearsal component; in this case, the transformation is from movements to sound. The sound produced by a vocal rehearsal is heard and remembered in auditory short-term memory.

In principle, although not in detail, the auditory scan is exactly analogous to the visual scan. The auditory scan selects some contents of auditory memory (e.g., the sound representation of one letter) and converts them into the address of a motor program. The address is remembered in the recognition buffer-memory, the program is executed by the rehearsal component, and the sounds are re-entered into auditory memory. By means of this rehearsal loop, information can be retained for a very long time in auditory short-term memory—many times longer than the decay time of the memory itself.

Perhaps in young children and some adults, the output of the rehearsal component must first enter into the outside world as sound before it can enter auditory memory, but most adults seem to have evolved a shortcut, which I have designated "subvocal rehearsal." In subvocal rehearsal, the subvocal output of the rehearsal component is entered into the auditory short-term memory just as though it had been a vocal output; i.e., auditory memory contains a memory of the sound of the letter. The rate of subvocal rehearsal can be measured,[6,10] and it is very interesting to note that it is identical with the rate of vocal rehearsal.

DISTINCTIONS BETWEEN SHORT- AND LONG-TERM MEMORY

Neural Distinctions

A short-term memory is a patch of neural tissue that is used over and over again for every appropriate input to the modality. For example, the retina undoubtedly serves as a short-term memory; a particular neuron in the retina might, by appropriate stimulus positioning, be activated by every letter that could be presented. But I suggest that the neurons involved in long-term memory are extremely specialized and are active only when their key is found. This does not mean that only one stimulus can activate a neuron in long-term memory, but rather that its range is infinitesimal, compared with the range of possible stimuli.

There is now fairly widespread agreement[1,9,12,18] that short-term memory is short-term not because its neurons remember poorly (although that is probably a factor) but because every new stimulus overwrites its predecessor or at least pushes it away from the fore of memory. Even silence or darkness, the absence of stimulation, is an input to short-term memory that must be recorded and that therefore inevitably drives out the record of previous stimulation.

Structural Distinctions

A short-term memory can be likened to a register in a computer; a long-term memory, to a section of core memory.[13] That is, a short-term memory is complicated and expensive (involving many neurons per unit of information stored), because the information in it is capable of being manipulated in many ways. For example, one bit of information can be compared with another bit of information, can be shifted, and so on. Every operation of this sort requires many connections. In computers, core memory is made as starkly economical as possible. So much is sacrificed to economy that no operation whatever (except perhaps erasure) is possible on the contents of long-term memory before they have been removed to a register. I propose that the same overriding principles that guided the evolution of computers to have a very few (but very intricate) registers and to have a great many (but very simple) core memory cells guided the evolution of nervous systems to have a few intricate short-term memories controlling great masses of long-term memory.

Functional and Behavioral Distinctions

The contents of short-term memory are retrieved by asking for the contents of the particular sensory memory, i.e., by giving the name of the memory. What did I just hear? What did I just see? The contents of long-term memory are retrieved by giving an association, i.e., a complex, highly specific input. For example, I say: "My telephone number is 582–2644. What is my telephone number?" You answer by asking yourself what was the last thing you heard. That it is Sperling's telephone number is irrelevant to the retrieval of the digits. However, if I meet you on the street tomorrow and ask you to repeat my telephone number, no short-term memory could possibly be equal to the job. You would need a memory that could be entered with the name "Sperling" (and perhaps some other concomitant bits of information) and that, when so prodded, would return the correct digits.

SIX LONG-TERM MEMORIES

Each of the active components in the model (Figure 1) is associated with a long-term memory. The long-term memory was constructed by the subject out of his past experience, long before his participation in any of my experiments. The three triangle components each use an intermodality long-term memory. The visual scan is served by an intermodality long-term memory that associates the address of the motor program for saying a letter with the visual features of that letter. The rehearsal component is served by a long-term memory that associates the auditory features of a sound with the motor program for producing that sound. The auditory scan is served by a long-term memory that associates the address of a motor program for producing a sound with the auditory features of that sound.

These intermodality long-term memories represent skills. As children, we learned to imitate sounds that we heard. We learned how to recognize letters, that is, to say the name of a letter when we saw it. Later, we learned how to read without speaking.

Beneath each short-term memory square in Figure 1 is a long-term memory of events within that modality. For example, long-term visual memory might contain the information necessary to recognize a particular face as familiar, even if no name or occasion can be associated with it. A preschool child would recognize some letters as familiar, even if he could not name them. Similarly, we have auditory memories of auditory events. Finally, we have the memory of the motor sequence necessary to say a letter.

The proper development of all six of these long-term memories is a prerequisite for the effective operation of the information-processing system outlined before.

Quantitative theories of short-term recall performance find it necessary to take into account a small amount of information that is getting into long-term memory from each trial and that, when there are repeated trials, significantly affects performance (see especially Atkinson and Shiffrin[1]). Although the experiments I have dealt with probably involve very little long-term memory (because each stimulus is viewed only once), it is obvious that something is entering the various long-term memories, at least occasionally.

I will concentrate now on the two aspects of the model that are of greatest relevance to reading: visual scanning and auditory memory.

VISUAL SCANNING

The Use of Visual Noise to Estimate Processing Rate

Brief visual exposures, by themselves, are useless for determining the rate at which visual information is processed. This is so because stimulus information persists in very-short-term visual memory for some undetermined time after the exposure, for at least 0.1 sec and usually for 0.2 sec or longer. If the duration of visual availability is undetermined, processing rate cannot be determined; duration of visual persistence and processing rate are complexly intermingled.

The way around this difficulty is to follow exposure of the stimulus letters by a "noise" postexposure field (Figure 2). The visual noise that I use looks like scattered bits and pieces of letters, and it effectively obliterates the visual persistence of the stimulus letters. By delaying the onset of the noise postexposure field, we allow the subject more time to scan the letters. Each 10–15 msec of delay enables him ultimately to report one additional letter, up to about three or four letters. This processing rate can be shown to be independent of the number of letters presented and of many other variations in procedure.

Serial or Parallel Processing?

In a brief exposure, are letters scanned one at a time, a new letter in each interval of 10–15 msec, or is information being gathered about several letters simultaneously at an overall rate equivalent to one new letter per 10–15 msec? A positive answer to the first question defines a serial scanning process, and to the second, a parallel process. I will go into greater depth in considering the problem of serial versus parallel processing, because it offers a good illustration of current research in information processing. The nonspecialist reader may have difficulty here, but I hope that he will persevere and obtain at least an appreciation of some contemporary methods and theories and of their potential power for studying the way in which words are read.

METHOD I

When I first confronted the serial-versus-parallel problem, I sought the answer by examining the rate at which information was acquired about each individual letter in a stimulus instead of looking only at the overall rate.[14] Subjects were presented with five random letters followed, after various intervals, by a noise postexposure field (Figure 2). Their task was to report correctly as many letters as they could, from all the locations. If they processed letters in a purely serial order, I would expect only the letter in the first location to be reported correctly at the briefest exposure; the first and second letter to be reported at longer exposures; then the first, second, and third; and so on. Let p_i be the

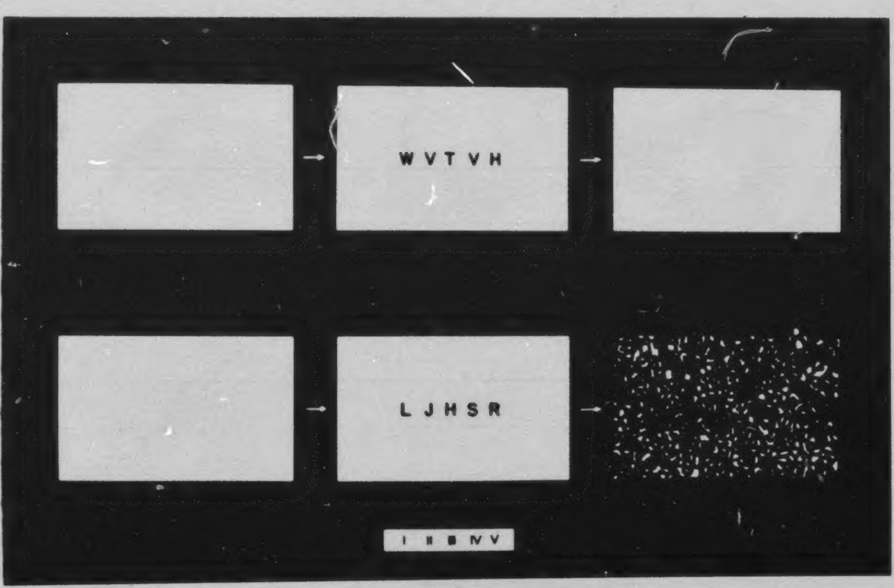

FIGURE 2 A normal tachistoscopic exposure sequence (top) and a postexposure visual noise sequence (bottom).

probability of correctly reporting the letter in the *i*th location. Considering each of the five letter-locations separately and plotting these p_i's as a function of exposure duration should yield a set of functions like those illustrated in Figure 3a. That is, the p_i functions in Figure 3a would be produced by a serial left-to-right scanning process whose overall theoretical performance best matches the observed performance. The first two letters are scanned quickly, the next two are scanned more slowly, and scanning of the last letter has hardly begun even at the longest exposure.

A purely parallel scanning process, in which information is retrieved at an equal rate from all five locations, would predict identical p_i at all locations (Figure 3b). Because all p_i's are the same, this p_i function also represents the observed overall percentage of correct responses.

The results of an actual test are shown in Figure 3c. The data illustrated are for one subject; tests of other subjects, including myself, yielded basically similar data. The downward concavity of all the observed p_i functions means that information is acquired, at each letter

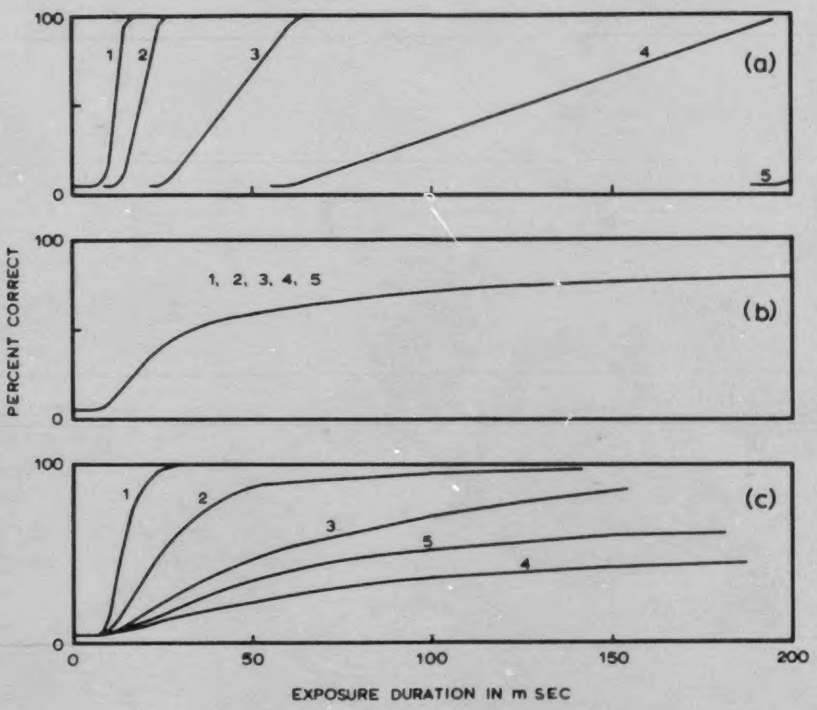

FIGURE 3 Accuracy of report of the letter at each location (1, . . . , 5) of a five-letter stimulus as a function of the exposure duration when exposure of the letters is followed by visual noise. (a) Theoretical data generated by a serial scan process with fixed order of scan. (b) Theoretical data generated by a parallel scan process having the same rate of information acquisition at all five locations. (c) Data of a typical subject (after Sperling[14]). These data are not corrected for chance guessing.

position, most rapidly immediately after the letter stimulus is turned on and that the rate diminishes as the exposure continues.* Information is acquired more rapidly at the first position than at the second, and so on, except that this subject acquired information more rapidly at the fifth position than at the fourth. Other subjects had different idiosyncratic orders.

* Percentage correct is a nonlinear (but monotonic) function of information retrieved. Plotting the results in terms of bits of information retrieved would exaggerate the concavity and strengthen the conclusion.

METHOD 2

Although the interpretation I have just given is stated in terms of parallel processing, one cannot rule out the possibility of some complex form of serial processing. To make a more sensitive test, more intricate stimulus sequences were required. Therefore, I gave up research for a year and worked at programming a computer to display visual stimuli on a cathode-ray oscilloscope.[3] The computer-produced demonstration that provides the strongest evidence of parallel processing is very similar to the procedure just described. Five letters are presented and followed by visual noise. The basic difference is that one of the letters is changed midway during its exposure (Figure 4). When this is done, for example in the fifth location, then almost invariably the first letter that appeared in that location is the one that is reported (that is, if the subject correctly reports anything at all from that location). This result with very brief exposures is just the opposite of the usual result when exposures are long (greater than 50 msec) or no postexposure noise field is used. In the latter circumstances, the second letter that occupies a location is the one that is reported.[2,5,11]

PREDICTIONS OF THE THEORIES OF SERIAL AND PARALLEL SCANNING

In a serial process, increasing exposure duration improves performance (increases p_i), because the ith location is more likely to have been scanned during a longer interval. Consider, for example, an exposure duration ΔT_1, which is just long enough so that $p_i = \Delta p$. Now con-

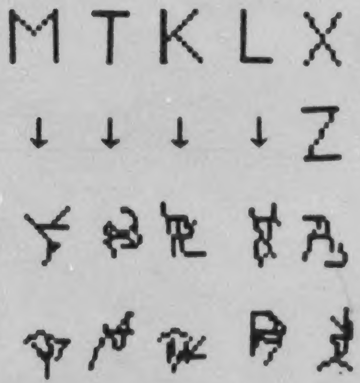

FIGURE 4 A computer-generated stimulus sequence for testing serial versus parallel processing. The initial stimulus is M T K L X. M; T, K, and L persist continuously until the onset of the postexposure visual noise; X is changed to Z in the middle of the exposure interval. Two consecutive noise fields are used to increase the effectiveness of the noise.

sider the additional exposure ΔT_2 that is needed to increase p_i to $2\Delta p$. In serial-scanning theory, an increase of Δp in p_i during ΔT_2 means that as many letter scans are made in ΔT_2 as in ΔT_1. If occasionally the *i*th position is scanned twice during the exposure, then more letter scans must be occurring in ΔT_2, inasmuch as occasionally a letter that was scanned in ΔT_1 will be rescanned in ΔT_2, and that would be a wasted scan. Serial-scanning theories can be characterized as basically "top-heavy." That is, when p_i is large—i.e., near the top of a graph like Figure 3b—then as many or more scanning attempts are needed to raise it by a given amount Δp, compared with the number when p_i is small.

Parallel-processing theory assumes that information is accumulated continuously. To increase p_i from 0.50 to 0.95, for example, requires less than one bit of information, whereas to increase p_i from 0.05 to 0.50 requires 3.3 bits (when there are 20 equiprobable stimulus letters). This example illustrates a general property of information-gathering systems: the first few bits of information change the probability of being correct only very slightly, and the last few bits cause big changes. Thus, parallel-processing theory is "bottom-heavy." The weighty processing occurs while p_i is small, i.e., near the bottom of Figure 3b.

To relate these theories to data, let us restrict ourselves, for the moment, to locations 3, 4, and 5, and to exposure durations of less than 100 msec. For example, consider an exposure of 50 msec and divide it, conceptually, into two consecutive intervals of 25 msec. Figure 3c shows that there is an equal or greater increase of p_i between 25 and 50 msec than between 0 and 25 msec in these three cases. Suppose now that at location 5 a different letter is presented in each of the two intervals—the experiment described above. According to the serial-scanning theory, an equal or greater amount of scanning occurs in the second interval, and so we would expect the second letter to be reported at least as often as the first letter.

In the parallel-scanning theory, in this instance, about 60% more information accumulates in the first 25 msec than in the second 25 msec, so we would expect the letter from the first interval to be reported more often. For parallel theory to predict quantitatively how much more often the first letter is reported than the second would require additional assumptions.

The experimental result was that the first letter is nearly always reported. We therefore reject the serial-scanning theory and tentatively accept the parallel-scanning theory. In 50 msec, the visual system achieves sufficient information, in parallel, from a letter array to recognize about three letters.

This conclusion is potentially important for understanding the reading of words. It means that the visual system has the capacity to process a word not merely letter by letter or by its overall shape, but as a complex pattern. Whether a word is recognized directly as a visual pattern, or the letters are recognized first and then the letter pattern is recognized as a word, or both processes occur together we do not yet know. But we do know now that the visual system has the capacity to gather enough information simultaneously–i.e., in parallel–from an array of letters (a word) to identify uniquely most ordinary words.

Extremely Rapid Visual Search in a Continuous Task

The experiments described above measured visual scanning speeds from single exposures only–that is, the speeds achieved in single bursts of scanning. Could subjects maintain the same high scanning speed in a continuous search task? The following experiment was devised to test this possibility. A computer[3] generates arrays of random letters and displays them on the cathode-ray oscilloscope. Figure 5 shows a sequence of 3 X 3 arrays. All the arrays except one consist entirely of random letters; the critical array contains the numeral "2" in a randomly selected location. The subject does not know in advance of the trial which array in the sequence will be the critical one, nor in which location the critical character will occur. His task is to look at the whole sequence of arrays and to say at which location the critical character has occurred. From the proportion of times the subject is

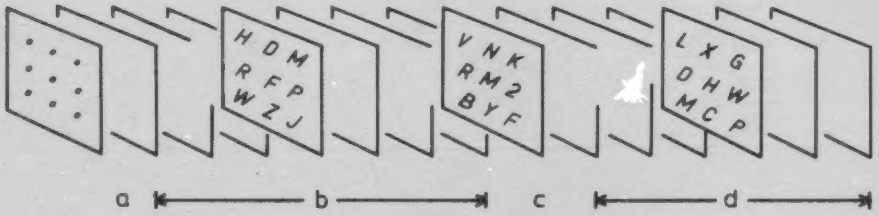

FIGURE 5 Diagram of the stimulus sequence in the sequential search procedure. a, fixation field. b, 6 to 12 letter arrays (randomly determined). c, the critical array, in this instance containing a "2" in the middle-right location. d, 12 more letter arrays.

able to make the correct response, we can deduce the speed with which he is able to scan characters to determine whether each is a "2." We have also trained a subject to detect the occurrence of any numeral among letters. The discrimination of an unknown one of ten numerals takes only slightly longer than the discrimination of a known single numeral.

We[16] have studied arrays containing from 1 to 25 letters, and presented new arrays at rates of 3 to 200 per second. We have not yet completed all these experiments, but the main results are already clear.

Subjects achieve the same high scanning speeds in the continuous-search procedure as were previously demonstrated for single bursts, 10–15 msec/letter. The highest scanning speeds are achieved at presentation rates of about 40 arrays per second with stimuli containing nine or more letters. Under these conditions, the fastest subject has broken solidly through the 10-msec barrier; he can scan characters for the absence of the numeral "5" faster than one letter per 8 msec. When nine-letter arrays are presented at a rate of 25 arrays per second (40 msec/array) he can identify the location of the critical character correctly about 70% of the time. That means that he is effectively monitoring five of the nine locations.* In terms of the parallel-scanning theory, this subject can process a fresh batch of five letters every 40 msec.

When the presentation rate is lowered, response accuracy improves, indicating that additional locations are being scanned. For example, my fast subject scans the equivalent of about 16 locations from a 25-letter array when new arrays are presented every 160 msec. His scanning speed goes down to about one letter per 10 msec at this rate, indicating that locations outside the most favored six are scanned more slowly. Sixteen positions are the maximum that he can scan in a brief exposure; lowering the rate does not improve his response accuracy. A more typical observer can scan three locations in 40 msec and a maximum of 10 locations in a single exposure.

In conception, these search experiments follow the pioneering work of Ulric Neisser,[7] who was the first to study rapid scanning of this kind. His subjects searched long lists for the presence of a critical item and signaled when they had found it. The important difference between our procedures is not that I use a detection method and he a reaction method, but that in Neisser's experiments the sequence of visual inputs is controlled by the subject's own eye movement, and in my experiments, by a computer. The optimal scanning rate in the searching for a "2" or a "5" occurs at presentation rates that are five times higher than the rate of eye movements. When the presentation rate of stimuli is lowered so that it is comparable with that of eye movements (e.g. 200–250 msec), then the processing capacity is virtually idle for the second half of the interval; it has done all or nearly all that it can do in the first half. With more complicated processing tasks, of course, processing times would be longer and the rate of eye movements might not be the limiting factor.

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*The estimate of the number of locations monitored depends somewhat on the guessing strategy that the subject is assumed to be using when he has not seen the critical character. If he could use absolutely the most efficient strategy, he could achieve a probability of being correct of 0.7 even when he monitored only 5.3 locations.

Although it is technically very difficult to implement, the method of searching sequentially presented displays is most promising for estimating processing times and will yield much of importance for reading. It already has provided one nontrivial conclusion: In simple search tasks, the limiting factor in performance is the rate at which eye movements can be made, and not the rate at which information can be processed.

AUDITORY SHORT-TERM MEMORY

Auditory Memory in Visual-Recall Tasks

I claim that the same factors limit recall of letters from brief visual exposures (assuming that the letters are clearly visible) and from auditory presentations, to such an extent that visual recall can be predicted from auditory recall.[17]

The original evidence of auditory components in visual-recall tasks was introspective (all subjects said they rehearsed subvocally) and indirect (subjects did not begin writing until a second or more after the exposure and their visual memory had decayed by then, so auditory memory was the only logical alternative).[10] The observation[15] that subjects suffered auditory confusion in visual recall (for example, D and 2 for T) was promising but not powerful. The powerful evidence comes from the measurement of "AS deficits," a technique that was introduced independently and almost simultaneously in three laboratories by Conrad, Wickelgren, and me (see Sperling and Speelman[17]), although it could and should have been invented 100 years earlier.

An AS deficit is defined as the decrement in performance caused by replacing a stimulus composed of acoustically different letters (for example, F, H, Q, and Y) with acoustically similar (AS) letters (for example, B, C, D, and G). The deficit technique can be applied to other dimensions, such as visual similarity, semantic similarity, and pronounceability. The main finding that concerns us here is that, in the usual test of visual recall, visual-similarity deficits are small, whereas AS deficits are large.[4] That auditory similarity should be a significant factor even in a task that involves only looking at letters and writing them—and never any overt auditory representation—is *prima facie* evidence of a role for auditory memory in visual-recall tasks.

To determine quantitatively how much of the memory load in visual-recall tasks is carried by auditory memory is more difficult. However, we[17] have been able to predict AS deficits in visual-recall tasks, in which subjects viewed a dozen letters exposed simultaneously, from the AS deficits of the same subjects in auditory tasks, in which they heard spoken lists of letters and were required to recall them. We could make these predictions from lists spoken at either one or two letters per second but not from lists spoken at rates of four letters per second. The rate of silent rehearsal was previously estimated to be three letters per second.[6] This rate seems to be critical for auditory presentations of random letters; at higher rates, recall performance deteriorates rapidly. I would conclude, pending evidence to the contrary, that the same factors limit recall from simultaneous visual presentations and limit recall of auditory sequences spoken at rates lower than four letters per second.

A Phonemic Model of Short-Term Auditory Memory

The results of 38 experimental conditions in which Mrs. Speelman and I measured recall of auditory stimuli could be predicted quite accurately from rules based on a phonemic model of short-term auditory memory.[1 (The predictions accounted for 0.96 of the variance of the data.) The phonemic model assumes (1) that individual phonemes are retained and forgotten independently in auditory memory; (2) that, when some of the constituent phonemes of a letter are forgotten, the letter is reconstructe as well as possible on the basis of the remaining phonemes; and (3) that, when the remaining phonemes do not suffice to identify the letter uniqu ly, a choice is made from among the most probable alternatives. Accordi to this theory, the reason that stimuli composed of letters chosen from A alphabets are poorly recalled is that they contain phonemes that do not help to discriminate among alternative members of the alphabets. For example, in the alphabet consisting of B, C, D, and G, the phoneme e is useless for discriminating among members, and retention of this phoneme in memory is a waste of space—a precisely predictable waste.

It is reasonable to call the memory into which an unrehearsed auditory stimulus enters an "auditory memory." Because the predictions of the model apply equally well to conditions in which there is little subvocal rehearsal and conditions in which there is a great deal of subvocal rehearsal, there is no need to postulate different memories for rehearsed and unrehearsed material. Finally, because the same generalizations govern recall of visual stimuli, there is no need to postulate a different memory for visual recall.

I should add that a really satisfactory paradigm for differentiating between the recognition buffer-memory and the auditory short-term memory has not yet been discovered. Therefore, when I say "auditory memory," I have to include in it the contribution of the rehearsal buffer-memory. That is not much of a complication, because, if the contribution of the recognition buffer is small, then it does not matter much, and if its contribution is large, then we can say that it must be very much like an auditory memory, in that the phonemic model (of auditory memory) accounts for so much of the evidence.

RECAPITULATION

A model of the processing of information from an array of letters has been proposed. It consists of the following components: a very-short-term, very-high-capacity visual memory; a visual scan component that converts the representation of a letter in visual memory into the address of the motor program for rehearsing the letter; a short-term memory for this address (recognition buffer-memory); a rehearsal component that converts the subvocal rehearsal into an auditory representation; an auditory short-term memory for the sound of the letter; and an auditory scan component that converts the auditory representation into the address of the motor program for rehearsing the letter.

Neural, functional, and behavioral criteria for distinguishing between short-term and long-term memory have also been proposed. A short-term memory is made up of neurons that are used over and over again by all inputs to the modality; complicated functions can be carried out on the contents of the memory; to retrieve the contents of memory requires knowledge only of the memory's name (i.e., the modality being served). The neurons that form a long-term memory are activated only by very specific inputs; no functions are carried out directly on the contents of memory; and the contents of memory can be retrieved only by means of very specific "associations." The components of the processing model are served by six kinds of long-term memory: visual, auditory, and motor long-term memories; and visual–motor, auditory–motor, and motor–auditory association long-term memories.

Experiments with visual postexposure noise fields are interpreted to mean that information is gathered simultaneously—i.e., in parallel—from three or more letter locations at an initial rate of one letter per 10–15 msec. The visual system thus has, in principle, the capacity to analyze a word not letter by letter nor by overall shape, but from information gathered, in parallel, from its component letters.

In the sequential-search procedure, a subject searches a computer-produced sequence of letter arrays for a character at an unknown location in one of them. The highest processing rate occurs when a new array occurs every 40 msec. This maximal rate of 25 arrays per second is 5 times the rate of eye movements. Lowering the sequence rate to the rate of eye movements grossly impairs search efficiency. The best subject was able to scan five locations every 40 msec and a maximum of about 16 locations (achieved in 160 msec) in a single brief exposure. It is concluded that, in simple visual-search tasks, the rate of eye movement will be a limiting factor in search rate.

The recall of visually presented arrays of letters is shown to suffer in a predictable way when acoustically similar letters (for instance, B, C, D, and G) are used. By comparing the recall of visually presented arrays with the recall of auditory letter sequences, it is concluded that visual letters are rehearsed at fewer than four letters per second (probably three per second) and that the rehearsal is stored in auditory short-term memory. Even when letter arrays are presented visually and are reported by writing (never overtly represented in an auditory mode), they are remembered in auditory short-term memory, as if they had been presented acoustically.

In this brief account, I have not considered how eye movements are controlled, how information from successive eye movements is integrated, how long-term memories are formed, or how subjects deal with words and bigger units of meaningful materials. These problems are relevant and important for the study of visual-information processing; some are considered elsewhere in these proceedings, but most, unfortunately, are far from solution.

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DISCUSSION

DR. KAGAN: Can I recall something you said a few minutes ago only because I have been rehearsing it? Would it not be stored in long-term memory?

DR. SPERLING: Certainly. Even very brief events often leave lasting memories; I wish I knew more about how and why. The stimulus materials in the experiments I have been discussing are random letters and numerals; they almost never get into long-term memory in just one trial. They can be recalled accurately for only a few seconds. To recall them after intervals of, say, 10 sec, a subject must rehearse them vocally or subvocally and must not be forced to accept any new information into his short-term memory. If either of these conditions is violated, the stimulus is forgotten.

Repeated rehearsal not only maintains the stimulus in short-term memory, but helps it to get into long-term memory. We do not know whether it is the act of rehearsing itself that is responsible, or whether it is merely that the longer a stimulus resides in short-term memory the likelier it is to enter long-term memory. To reiterate, the essence of short-term memory is that the same patch of neural tissue is used over and over again by new inputs. Obviously, this same tissue cannot also serve as a long-term memory.

DR. ULLMAN: Is the use of short-term memory a prerequisite for the formation of long-term memories?

DR. SPERLING: I would say that visual inputs pass through visual short-term memory, and auditory inputs pass through auditory short-term memory. Given the complexity of long-term memory, I would be rash to venture beyond that simple statement.

DR. SCHUBERT: The leaders in the field of reading would have us believe that some children are visually minded and some children are kinesthetically minded. When you say that your subjects rehearse subvocally and you relate their performance to this kind of rehearsal, are you referring in particular to kinesthetically minded subjects?

DR. SPERLING: No. What I am saying is that, in the particular recall tasks that we have devised with random-letter stimulus materials, auditory memory is so much more effective than visual that we barely detect an effect of visual memory. If we were dealing with words and language, or with pictures, it might be quite different. Incidentally, Dr. Michael Siegal is using our acoustically similar stimuli for memory tests on children with eidetic imagery, and finds that even these extremely visually competent subjects do not behave differently from subjects *on these tasks.*

DR. HOCHBERG: Can you predict one kind of memory from the other?

DR. SPERLING: No. I did not say that I could predict the capacity of a subject's visual memory from the capacity of his auditory memory, but rather that I could predict a subject's performance on the recall of visual stimuli from his performance on the recall of auditory stimuli. The reason is that the stimuli that Mrs. Speelman and I used are remembered in auditory memory even if they are presented visually. That is, when we make this assumption, we can predict performance.

I do not wish to be put into the position of saying that there is no visual memory; there certainly is. But except for the very-short-term visual memory, visual memory seems to be basically unadapted to recall, and so we do not find much evidence of it in recall tests. To find out about short-term visual memory, or perhaps intermediate-term visual memory, we have to use recognition procedures. Even that is not sufficient in itself. If efficient verbal codes exist, they will be remembered in auditory memory and in other memories and thereby override the visual phenomena that we are trying to measure. The stimuli to be recognized visually have to be made nonverbalizable. Or they have to be so constructed that a verbal description of them would be so inefficient that subjects would not be tempted to try it or, if they did, it would not aid them. I use a computer to generate visual stimuli and, with small modifications in the program (occasionally unintentional), it produces good characters for a recognition experiment. These are made of basically the same segments as letters, but joined in different ways. They look like elements from an unfamiliar Eastern scrawl (see Figure 4). The computer produces an almost limitless variety of different characters, so that none of them becomes familiar.

In our tests, we show the subject a stimulus twice, with an interval of a few milliseconds to 16 sec between the two presentations. The stimulus is composed of six or ten of these characters. In the second presentation, one of the characters is altered, and the subject's task is to say which character. From the accuracy of his response, we deduce how many characters he is remembering correctly. In preliminary experiments with this method, we again found the very-short-term, very-high-capacity visual memory. Beyond the first quarter of a second, performance was disappointingly poor. Subjects are able to retain enough information about only two or three characters to recognize that they have been changed. However, the time constant of forgetting was, surprisingly, so long that I could not estimate it properly. These experiments, like most others that have been used to investigate visual memory, have their problems (Hochberg, J., in R. N. Haber, Ed. Contemporary Research and Theory in Visual Perception. New York: Holt, Rinehart and Winston, Inc., 1968. pp. 309–331), but I cite them to show that measurements of short-term visual memory are being made (see also Shepard, R. N., J. Verbal Learning Verbal Behav. 6:156–163, 1967).

DR. SHANKWEILER: It seems to me that you should not attribute your findings to auditory memory. I suggest that subjects are coding into language.

DR. SPERLING: The kind of auditory memory I have been discussing is basically very simple, although some of its properties are very complex and may surprise us. If you had available a pile of neurons, I could tell you how to connect them to make an auditory memory. In conception, it is very much like a sound spectrograph; the same basic construction would serve either a mouse or a man. It is a memory for sounds; let us reach semantic agreement on that point.

To construct a memory that remembers not merely sounds but linguistic units would be incredibly more difficult. I should think that one would not even undertake it unless one already had a very good auditory memory for sounds. But that is a philosophic answer. That I like to keep things simple does not mean that nature does. In fact, your hypothesis about linguistic memory probably could be formulated specifically in terms of an alternative model and subjected to experimental investigation. I invite you to do so.

JULIAN HOCHBERG

Attention in Perception and Reading

Reading text, listening to speech, viewing scenes and pictures—these activities are not automatic responses to an array or sequence of patterned stimuli. A reader does not simply look at a block of text and absorb its message. He must "pay attention" to the display; what this means has not yet been well worked out. Attention is often thought of as a separate faculty that operates on the perceptual process—e.g., as a determinant of degree of arousal or sensory facilitation or as a gate or a filter. If attention functions in this general way, it might be of practical importance to study its effects on reading behavior, but it would not be very interesting, theoretically, as a source of insight into the nature of the reading process in particular, or of perceptual processes in general.

Alternatively, one can consider that the phenomena of attention are intrinsic and inseparable aspects of the perceptual process. One might think of statements, going back to Brentano,[1] that perception is purposive, intentional, and directed, and so on—statements that have to be fleshed out if they are to be meaningful. Let us view the reading process as an intentional activity: an activity that has unique characteristics, but that also draws on abilities used in listening to speech, on the one hand, and in looking at objects and pictures, on the other.

LISTENING TO SPEECH

Consider the act of listening to speech: It is easy to demonstrate that attention is necessary to speech perception. As a considerable amount of research has shown, if a subject is required to attend to one of two simultaneous and fairly rapid monologues, he seems to fail to hear the content of the unattended message. Many workers have explained such selective attention by positing a filter that passes the attended signals and attenuates or even blocks the unattended. Such a filter would require many unlikely and complex properties. If both voices speak the same message, but the unattended one lags behind the attended one, after a while most subjects realize that both messages are the same.[6] However, as a general statement, it is not true that the content of the unattended message is unheard.

If, for example, the subject's own name appears in the unattended channel, he will pick it up, and, although it is easy to see how one could "tune" a filter in terms of frequency or any other simple characteristic, it is very hard to see how one might do so in terms of analyzed verbal meaning. For these and other reasons, Neisser[5] and I[2,3] have proposed that there is no filter. Suppose, instead, that the listener does the following when he receives a phoneme in a voice to which he wants to attend. He selects a plan to produce some well-practiced fragment of speech that starts with the phoneme just received. By "well-practiced fragment of speech," I do not mean that he is actually pacing–actually going through a subvocalization–nor that auditory images are going through his mind. I mean that he has readied a sensorimotor program that would, if activated, result in verbal articulation. He selects a well-practiced fragment of speech that starts with the phoneme that he has just received and listens for the later occurrence of one or two distinctive phonemes in the speech fragments. If he actually receives what he anticipates, he goes on to anticipate the next speech fragment. Thus, it is the expectations that are being tested, rather than the entire sequence of phonemes that were presented. What is important is the confirmed speech fragments–the listener's expectations, rather than the soundwaves actually presented.

This would explain what happens in the two-channel experiment. The subject makes an active anticipatory response to the initial phonemes that he hears in the voice to which he is to attend. Meanwhile, phonemes that are uttered by the unattended voice are briefly stored as unrelated sounds, not as the confirmed expectations, and will generally fade from memory while the subject reports the attended message. The unattended message is presented but not anticipated and encoded in any memorable form. The assumption that attentive listening depends on this kind of linguistic extrapolation yields several predictions about two-channel listening experiments; in general, these predictions seem to fit the existing data.

The most frequent technique used is shadowing. This procedure ensures that attention is being paid to the desired channel, and at the same time one has a measure of how well that channel is being heard. If the prime message does not have any syntactic redundancy, but is a group of unrelated sounds that have no meaning, then extrapolation is impossible—and so is shadowing. This observation makes good sense in terms of an expectancy model of attention, but it makes little sense in terms of a filter.

Of course, there may also be a filter, and there are other kinds of intellectual capabilities that may contribute to attention in general. But my point is that, if learning to listen to speech is a process involving redundancies of sound, then the two-channel attentional phenomena result from an intrinsic aspect of the listening process and not from the action of an additional faculty. Something like this should also occur in reading, which is rooted in the listening and speaking process.

LOOKING AT TEXT

Reading must draw also on the abilities of the visual–motor system, whose skills are originally developed in looking at the world of scenes and objects. Like listening, looking brings the subject a temporal sequence of patterned stimuli. He must integrate the successive, restricted glimpses of the world that he obtains about four times a second through his small area of clear foveal vision. Unlike the listener, however, the viewer does not have to rely only on contextual redundancy to anticipate the next moment's stimuli. His wide area of peripheral vision gives him an intimation of the future, of what will meet his next glance. And, because eye movements are fully programmed in advance of their execution, any efficient sampling of the peripheral vision also tells him roughly where his present fixation fits in the overall pattern.

Obviously, there are many ways to retrieve meaning from a printed page or a scene, and, as Alpern noted earlier (p. 119), the periphery is not essential to vision. You can read without it, and, as Glickstein told us (p. 139), you can retain relatively little of the nervous system and still have good form perception. I think such facts are misleading; I think it is more a question of how the visual equipment is used when you do have it than a question of what kind of behavior process or prosthetic capability you can call on when you do not have normal equipment.

Two processes are available to vision, and consequently to reading, that make it different from listening: We can, to some extent, anticipate what is coming on the basis of what is on the retina in peripheral vision, and we also, to some extent, have a record of what we saw after fixation from what is still present in peripheral vision. However, experiments in which the subject views a moving scène without using peripheral vision show that the adult observer has cognitive bases, in addition to those offered by peripheral vision, for expecting what each new fixation will bring. He has cognitive schemata into which he can fit each glimpse, so that he has a single "map" to store during the looking period and not a sequence of individual glimpses.

Reading is a form of looking, and, as a kind of visual perceptual behavior, it must share some of the characteristics of looking. But the attentional components of normal looking at scenes and shapes, which are undoubtedly well practiced long before school age, probably run counter to those needed when a person is learning to read or is reading a difficult text. There are few occasions in normal looking to make small, successive adjacent fixations, whereas the first task for a child when he learns to read is to put letters together into words by such adjacent fixations--surely an unaccustomed task for the visual-motor system.

A practiced reader, in contrast, has freed himself from that unpleasant necessity. He samples a display of the text, rather than looking at each letter. He has learned to respond effectively to the few features seen with clear foveal vision by expecting an entire word or even a phrase. He needs to fixate only the parts of the array farther along the page that will enable him to formulate new guesses as well as to check his current guesses. His expectations of what he will find are based on the syntax and the meaning of what he has just read, and they must also be based on the blurred view provided by peripheral vision. A beginning reader probably makes little use of peripheral information, and is therefore less hampered than an experienced reader when peripheral vision—peripheral information—is reduced, for example, by making the interword spaces indiscriminable to peripheral vision by running the words together.[4] Presumably, the same is true of a reader's ability to guide his sampling of the text by cognitive factors. The better the reader, the more widespread the fixations by which he samples the text, as long as the text provides contextual redundancy and as long as the task permits the reader to leave individual letters uninspected—that is, as long as the reader attends to meaning or content, rather than to spelling.

Like the listener, therefore, the reader is engaged in formulating and testing speech fragments, but he can use the information given in peripheral vision (as informed by his linguistic expectancies) to select the places at which he obtains successive stimulus input. This is like running very fast over broken terrain; anticipation is needed for making adjustments. This is a headlong flight through the text, not "information processing," letter by letter.

For example, when a reader follows a line of type, letters above and below it are often technically visible and fall well within his acuity. I suggest that, like the voice in the unattended channel previously described, letters above and below the line are simply not anticipated, not encoded, and therefore not remembered in any linguistic structure. And, as with two-channel listening experiments, it should be possible to produce intrusions into normal linear reading by placing suitable material, such as a subject's own name, in the unattended lines. This is a prediction that I made earlier[3] and that was recently confirmed in an unpublished report by Neisser.

This brings me back to the question: What would happen if you presented bits of text to a subject in a sequence that would be similar to views provided by reading saccades but not actually produced by saccades initiated by the subject? Would the result be normal reading, inasmuch as the stimulus sequence is similar to that which would result from an intention to read? I argue that it would not—that, when the subject moves his eyes in reading, he is not just moving his eyes, but is looking for specific features, testing his expectations of what he will see next. If he is simply receiving bits of text, with no attempt to formulate and test linguistic structures, the letters or words that he sees should quickly exceed his memory span. The display should provide neither meaning nor clear temporal organization. In contrast, when he looks at text with an intention to read it, he fits speech fragments to the letters glimpsed with each fixation; the speech fragments then afford a meaningful linguistic structure by which successive glimpses may be stored and repeated.

In this reading model, "paying attention" is an integral part of the reading process, and it depends on the task and on the reader's syntactic and semantic knowledge and vocabulary. Some of the implications have been tested in research; many more have not.

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DISCUSSION

DR. LISS: Speech is different from reading, in that you are working on what you just heard, rather than anticipating. For example, if I said–suppose you had wanted to–now, I am saying–

DR. HOCHBERG: I was trying to listen to you, and what I heard was, of course, impossible to anticipate. My presentation is obviously simplified, but I think that your intuitive objection is also too simple. That no skilled action requires attentional activity is speculation on your part. If you are listening to difficult speech–relatively low-redundancy speech–coming at a fairly rapid clip, of course your anticipations are relatively slow. If you had been able to attend to what I was saying very, very carefully, listened for all the inflections, it would have been another matter.

DR. LISS: You are reducing the possible alternatives to such a low level that you can anticipate full names. Usually, we do not speak this carefully, and the senses are receiving novel stimuli and are unable to differentiate sentences. There are too many possibilities to conclude that.

DR. HOCHBERG: The sentences may be novel, but the contexts in which those sentences are used under normal conditions are far from novel. I am sure you did not have to pay attention to full names to get a full name. You do not have to test those full names. These are working names, and you have far from novel characteristics here—you can hear the names with these preliminary sounds just as well as with the full names. Just listen to all the political speeches. I think you will find that the only information coming in that you actually *need* may be the first few sounds, to give a clue as to what the speaker is saying. You are perfectly capable of handling familiar speech at much greater speeds than you normally would be able to tolerate with denser, more foreign types of speech.

One does something like that in reading, except that the reader can control his own rate of reading. And the span of anticipation in reading will also depend on both task and redundancy. If you are actually reading a paper, you are not attending to the meaning of what is being said around you. Also, you can read a paper in the sense that you are only proofreading; you might not be able to explain it or make any sense out of what you are reading, but still be doing a good job of proofreading.

It seems to me that you rather nicely focused on the problem of the child who starts to read for the first time. As to whether what I have said has any implication for the varying methods of teaching reading, I do not know. I have a couple of disjointed thoughts about that. One of them is about the old prescription that seems to be universally followed of using large, well-spaced type with very few words on a page. This can have many explanations. An obvious one is that it requires the beginning reader to hobble his eye movements less; he has to make fewer small, adjacent saccades that run counter to his normal scene-sampling strategies. Also, you can say that the child is not capable of discriminating small letters. I do not know what that means in any reasonable sense, inasmuch as his visual acuity is probably a lot better than mine when he starts to read. It seems clear to me that limitations in his sampling behavior are involved, not anything about receptive fields or about vision itself.

It is by no means rare for children to teach themselves to read. A very small, unknown proportion of children do, in fact, teach themselves to read. This seems to me to be such an important fact concerning the alleged difficulty of acquiring reading capacities that it ought not to be ignored. How these children teach themselves to read, and by what routes, should be attended to. I find it implausible that they are all following the same route. I expect that it does not matter how they get through the initial stages, how they learn to sample, how to make good guesses. To get back to the point of this meeting, reading, let us try this prescription: The child should be started with material as close to his own listening vocabulary—the units into which he normally breaks heard speech—as possible. If you are dealing with children of the ghetto, whose habitual language is very different from that of the initial primer, then you should design primers in which the constraints are close to the guesses that he is going to make when looking at a block of letters. Primers should be designed so that the child's guesses are going to be right more often than not. Letter-group combinations that do not encourage guessing are probably nonreinforcing. Certain things start children on skimming, rather than reading, and there is a distinction between skimming and reading that workers on "reading itself" have made that is probably unwise.

DR. MASLAND: Some children have much more difficulty with auditory stimuli, particularly verbal, than other stimuli. It is not important to have auditory attention, which is involved, as you and others have pointed out, in the visual representation of language; but it is important to be able to use methods that help children increase their auditory attention when the meanings of the things said are presented in a visual form.

DR. HOCHBERG: I was speaking to just that point. If the child has a vocabulary of expectancies you can increase the span of auditory attention simply by increasing the chunk size of the message, that is, the chunk size for him. Obviously, if someone says "Fourscore and seven years ago," I only have to check it minutes hence to see whether or not it is the Gettysburg Address, although I am not really attending in between. And that is why, at the beginning of my presentation, I put "pay attention" in quotes. Among children who have difficulty in paying attention, there may be some for whom the difficulty is organic; there may be some for whom this is proof of various motivational factors—they just do not want to listen; and there may be some who cannot pay attention because the redundancy is too low. For the latter to "pay attention" would require far more predictive ability and far more alertness than could be mustered, far shorter reaction times, far larger vocabulary. I think that these elements would have to be separated.

DR. HIRSH: I wonder whether the vision people can tell us whether the span, measured in angles or distances, can vary as one reads across a line? This, it seems to me, would be necessary to make reading analogous to listening to speech. We are talking about the problem of segmentation; in the case of listening to spoken language, the size of the chunk that you take in varies from point to point in a sentence.

I am sure we would all agree that the visual process of reading would be impossible if we forced the child to read through a reduction tube only one letter big, so that he had to scan one letter at a time. However, if language does make this great difference in reading, then it ought to be true that glances or their content on the retina are of different sizes.

DR. HOCHBERG: You cannot ask that question in that form, I think, because the acuity needed to pick up the confirmatory information for any given message is going to be a function of the redundancy of that message to begin with. If my hypothesis about how much information is going to come next is going to be answered by "Yes, here's a space and a period," I can pick that up at the periphery, maybe 10 deg out. If I am going to have to distinguish between an "e" and an "s," or an "a" and an "s," it is going to have to be, say, within 5 deg. So, indeed, there is a variable span already given in terms of the kind of question that you have asked. Now, if you could partial out that redundancy factor, then you might ask the vision people whether the span is variable, and the answer would probably be "no." But I do not see how you can partial out that redundancy.

DR. SPERLING: It has been shown that, in a single glance, a person usually gets far more information than he can process before making the next glance. It is useful, therefore, to distinguish between the amount of information available in a glance and the fraction of that information that is ultimately processed.

DR. MASLAND: I would like to ask for clarification of a problem that seems to represent a very important concept. You mentioned that, in regard to visual attention, a person samples his surroundings against a relatively fixed map. That is to say, a spatial display is being continually sampled, but it is, in a sense, a fixed pattern, whereas the auditory display and the sampling of it are temporally dispersed elements. This represents an important aspect of reading tests, which you skimmed over rather lightly: a person in a sampling has a visual map fixed in such a fashion as to achieve a sequential event or a series of events that fits into an auditory display, which is a temporal display.

DR. HOCHBERG: You are correct in your interpretation, and you are correct that I did skim over it lightly. You are also correct in the implication that there is not a lot of filling-in that I can do except to say that it is indeed a problem and that the ability to integrate our successive glimpses of the world into fixed spatial perceptual maps must be rather well established before we learn to read. That ability is certainly drawn on in the reading process, for example, when you go from one line to the next on a printed page as that page moves around while you read in a jogging trolley car.

But the temporal sequence, I have been arguing, is not essentially a visual function; it is a linguistic function. It is a matter of simple convention that you are always going to read from left to right until you get to the end of the line. That alone is not sufficient to impose a constant perceptual order; it can only guide a sequence of looking. The temporal order is given by the linguistic structure and is not part of the visual process in any direct sense.

DR. MASLAND: It is unproved that the spatial map is primarily a righthanded sphere or preoccupation, if you will, and the auditory temporal map primarily a lefthanded sphere. I do not know whether Dr. Sperry's observations confirm that.

DR. INGRAM: Dr. Hochberg has emphasized the importance of the child's learning about the segmental features and the syntax of the spoken language he is in the process of acquiring. But he has not emphasized the importance of nonsegmental features. Rhythm and intonational patterns may well be crucial to the child trying to comprehend the significance of what is said to him at an early stage of speech development. Rules of grammar are probably acquired considerably later. Consider, for example, the complexity of sentences involving the use of the negative, such as "He is coming, isn't he?" or "He isn't coming, is he?" Children have to learn how to use the negative in these situations. It is wrong, for example, to say "He is not coming, isn't he?" or "He is coming, is he?" Before he reaches an understanding of the grammatical rules underlying such utterances, he depends on other informational aspects of language.

DR. HOCHBERG: "He is coming, isn't he?"—there are intonations, paralinguistics, what the story is about, and so on. That is probably why children's books are all picture books—as a substitute for such sources of information.

DR. LINDSLEY: I would like to try to tie together something Dr. Hochberg has said with what we have heard with respect to electric potentials of the brain.

I think that we are all aware that one can look and not see, one can see and not perceive, and one can perceive and not remember. Perhaps one can perceive and remember and not learn or develop a concept. Dr. Hochberg was emphasizing, particularly, selective attention to specific points, perhaps to the type that one reads, or perhaps to something else that is related to it. In talking about electric potentials of the brain, Dr. Buser mentioned associative potentials that have a longer latency than primary potentials. In our work on selective attentiveness (Spong, Haider, and Lindsley, Science 148:395-397, 1965), we have given subjects a pattern of stimuli made up of clicks and flashes alternating in sequence and have instructed them to pay attention to flashes and ignore clicks. The average evoked potentials to the flashes, recorded over the visual area of the brain, were enhanced, whereas the responses to the same flashes were reduced when the subjects were instructed to pay attention to clicks and ignore flashes. The potentials that were enhanced in amplitude were the late components of the evoked response, which would correspond roughly in latency to some of the late or association area responses described by Dr. Buser.

The question was raised for Dr. Buser whether shorter-latency potentials from primary receiving areas spread to association areas, where they are recorded with longer latency. Apparently, they are relatively independent and represent different systems, inasmuch as Thompson, Johnson, and Hoopes (J. Neurophysiol. 26: 343-364, 1963) found short-latency, modality-specific responses over primary sensory areas, whereas the longer-latency responses of more than one sense mode were recorded from association areas, which seemed to be convergence centers. These responses, even in different association or convergence centers, tended to be highly correlated in their response amplitude, in contrast with the modality-specific responses recorded in the specific sensory zones of the cortex, which showed differential response characteristics. In our work with human subjects, recording average evoked potentials to visual, auditory, and somatosensory (median nerve) stimulation, we asked subjects to pay attention to the stimuli of one mode and ignore the other two. The primary, short-latency components of the response to somatosensory stimulation were reduced in amplitude when that sensory mode was selectively attended to, whereas the longer-latency components of the response were enhanced. Thus, the short-latency or primary components of the response appear to represent one system, and the longer-latency components, another, in view of their differential reaction to the same stimuli under the same attentive set or condition (Spong and Lindsley, unpublished data).

We have found that the degree of amplitude enhancement of these late potentials, during selective attention, is a function also of the general arousal or activation level that one can create by producing a more difficult task to perform; therefore, the two things seem to go hand in hand. There is a selective attention effect and an arousal effect. In learning to read, some kind of general arousal or activation or learning level would be helpful; presumably, it would be generated by the motivating influences to read that one could have. The specific, selective attention factors are, as Dr. Hochberg pointed out, partly anticipation. I would agree with Dr. Kagan that they may stem from what has just occurred in the past, because that may serve as a guide or anticipation or expectancy of what is to come in the future.

I would like to emphasize that associated with this there is also an electric potential different from that just discussed. This is what Walter *et al.* (Nature 203:380–384, 1964) have called a "contingent negative variation" (CNV), or a slow, negative, d-c potential shift. In their experimental situation, the subject was given a warning sound and told that flashes of light would follow and that he should press a key quickly to stop them when they occurred. When the sound comes, and before the flashes appear and before the key is pressed, there is a buildup of a slow negative potential, a d-c shift that discharges when the anticipated flashes occur. We have been working on a similar experiment with a little more relevance to this particular problem. We presented three flashes 0.5 sec apart, and the subject was instructed to press a key on the third flash. Then we practiced the subject on another stimulus sequence, with the third flash delayed 1 sec after the second one. When the subject knew that he was going to get one or the other pattern, and there was no uncertainty about it and no probability decision to make, there were evoked potentials to each flash, but no CNV or d-c potential shift building up to the onset of the third flash. Thus, this expectancy wave or potential shift seems to reflect anticipation or probability decision on the part of the subject; but, unless there is uncertainty associated with it, the CNV does not occur.

I think that, in the reading situation, the extent to which we are interested in reading depends on the extent to which we can anticipate, as Dr. Hochberg said, or on what expectancy we have concerning what is to come, either because we want to confirm something (if it is, say, scientific writing) or because it is something that we can anticipate as new and unique. This can be contrasted with proofreading, in which one is simply looking to see whether the words are spelled or put together correctly and does not necessarily understand or remember the content of what is read. In other words, the purpose or goal of the reading makes a great deal of difference. When you read for your own information, you read for what you expect to get out of it, and not in terms of these other characteristics.

DR. KAGAN: I was under the impression that you only get this rising potential if the subject has to make a motor act, and not under such circumstances as when you say, "When the light comes on, you are going to see an extremely attractive picture." Is it correct that you need a motor signal?

DR. LINDSLEY: I do not believe so. Vaughan, Kosta, and Ritter (Electroenceph. Clin. Neurophysiol. 25:1-10, 1968) have published an article that suggests that, but I think we have evidence that the motor response is not needed. In our experiment, there is no motor response.

DR. KAGAN: The question is, do you have to have an intention to make a motor response to get the effect?

DR. LINDSLEY: I do not think so, but it is still debatable.

IRA J. HIRSH

Visual and Auditory Perception and Language Learning

It is assumed that there is a relationship between language comprehension and reading. One of the reasons it would be so difficult to teach a 1-year-old child to read is that there is very little written material in 1-year-old language. Perceptual modalities have something to do with learning a language, so that we really want to hear from the reading experts about how language affects reading. I will add a little information on that point myself, and then comment on one aspect of language learning with particular reference to visual and auditory perception. Finally, I would like to suggest some relationships between perception and response, especially for speech.

LANGUAGE LEARNING

Children from the age of about a year show evidence of both producing and appreciating grammatical structure, using phrases and sentences, although the rules that they appear to follow may not happen to be the adult rules. Until about 3 or 3½ years of age, they seem to learn, not by memorizing cases, declensions, inflections, and conjugations, but rather by comprehending what some linguists have called the "deep structure" that is common to a variety of surface grammatical forms. Vocabulary does not appear to become asymptotic until about the twenties, at least in most people.

I would like to concentrate on a third aspect of language, its phonology, because that is the spoken form of the language that is represented in the written form. Phonetic development takes place after the age of about 6 months. Before that, there is some babbling or noisemaking that seems to be devoid of recognizable phonetic content. After about 6 months, we begin to hear some phoneme-like production. Beginning at about a year, these speech-like sounds are used in phrases and sentences.

PHONOLOGY

One of the methods of teaching reading tries to establish a code-like relation between printed symbols and spoken ones. Unfortunately (in some cases), the printed-symbol unit is the letter, and the spoken-symbol unit is the phoneme. It might be dangerous to use phonemes as models of printed letters, for two reasons. First, phonemes do not exist as entities except in linguistic analysis. The speech output of a talker is essentially continuous sound. It is broken up into chunks, but by listeners who know the speaker's language. This chunking or segmenting is an active process on the listeners' part. In reading, the chunks or words are already marked off—by spaces. Second, the analyzed phonemes, as products of analysis, are themselves not very consistent. I would like to presen some data that Eguchi and I will publish soon.[1]

Spectral Features

Spectrographic analysis displays frequencies of sound on the ordinate of a graph, and can be read essentially as one reads music; pitch or frequencies go up or down, and time is shown on the abscissa. By connecting the amplitude peaks, we outline the "formants" characteristic of that vowel.

We asked some children, ranging in age from 3 to 13 years, and some adults to speak two sentences: "He has a blue pen" and "I am tall." We had them repeat these sentences on five different occasions, and analyzed all such productions with the spectrographic technique. We were particularly interested in how precise the subjects were in saying the same thing five times. If we characterize the vowel in "he" as reaching a first peak or formant in one region of the spectrum and a second formant in another region, how repeatable are those formants? The repeatability under study here is measured by the intertrial standard deviation (SD) of those formants for each person, as shown in Figure 1. The points show an average SD for each age group.

The standard deviations for each subject are based on five recitations of the two sentences. The points represent the vowel sounds in the words of the sentences. Variances for the first formant in each vowel are different for the different vowels.

I think the impression is clear for the first formant. Children are very variable when they are 3 or 4 years old, and their speech variability decreases as their precision of speech increases, until they reach the age of about 11 or 12. At that time we can speak of speech "habits" having been formed, but not before.

Figure 2 shows similar results for the second formant. There is no remarkable exception to the rule that variabilities decrease over time.

What worries me about teaching youngsters that a certain letter represents a certain sound is that they do not produce a particular sound each time they intend a phoneme. One of the reasons this study got started was the difficulty we had when we moved to another country and spoke a second language. I have great difficulty in understanding French youngsters, although I understand French adults well. My Japanese colleague finds it impossible to understand American children, but he has little trouble with American adults. The reason is that these productions are extremely variable in children. We must conclude that the phoneme is, at best, a very labile model to use for the printed letter.

Temporal Features

The vowel spectrum for the words "blue" and "tall" is characteristic of a vowel in a steady state for some period. But there are also, in speech, some features of phonemes that depend on temporal gaps. We can lump these together under the term "temporal features."

The spoken names of letters (as in the alphabet) do not always correspond to the sounds of the spoken letters themselves, and this disparity is particularly great in cases in which one cannot even say what a letter stands for, except as a transition between two other sounds.

We looked at one such temporal feature: the interval between the "b"

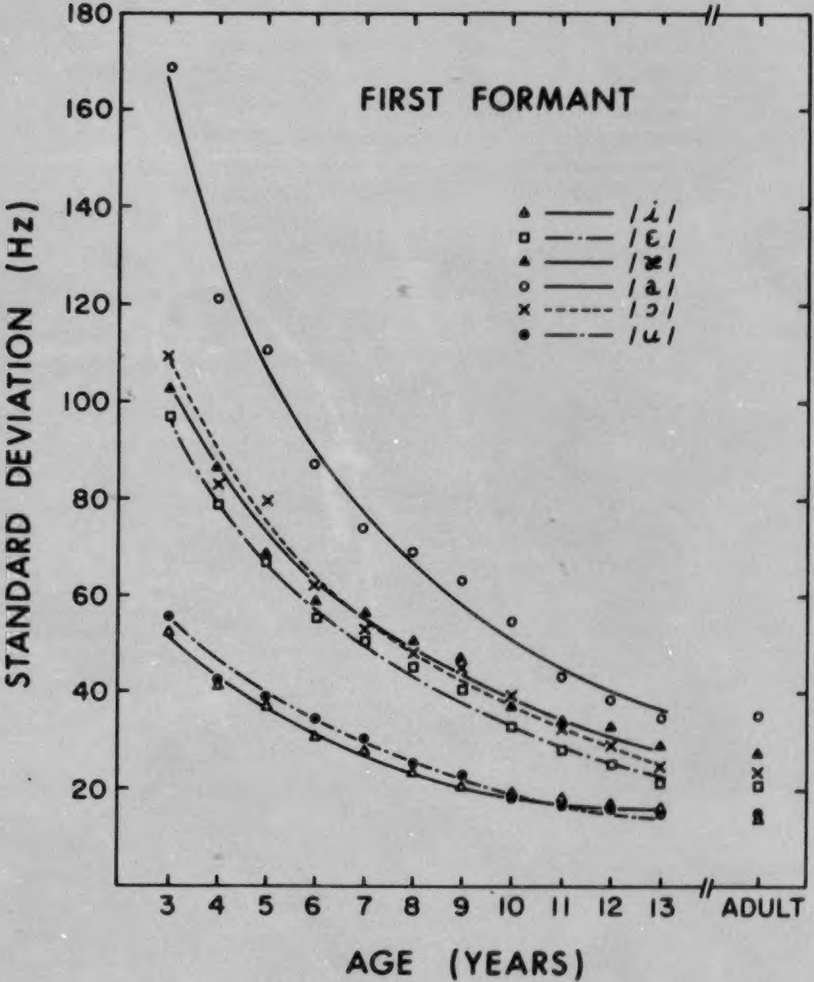

FIGURE 1 Intrasubject variability in formant 1 typical of the different age groups, as a function of age. Each point represents the square root of the average variance for each age group. (Reprinted with permission from Eguchi and Hirsh.[1])

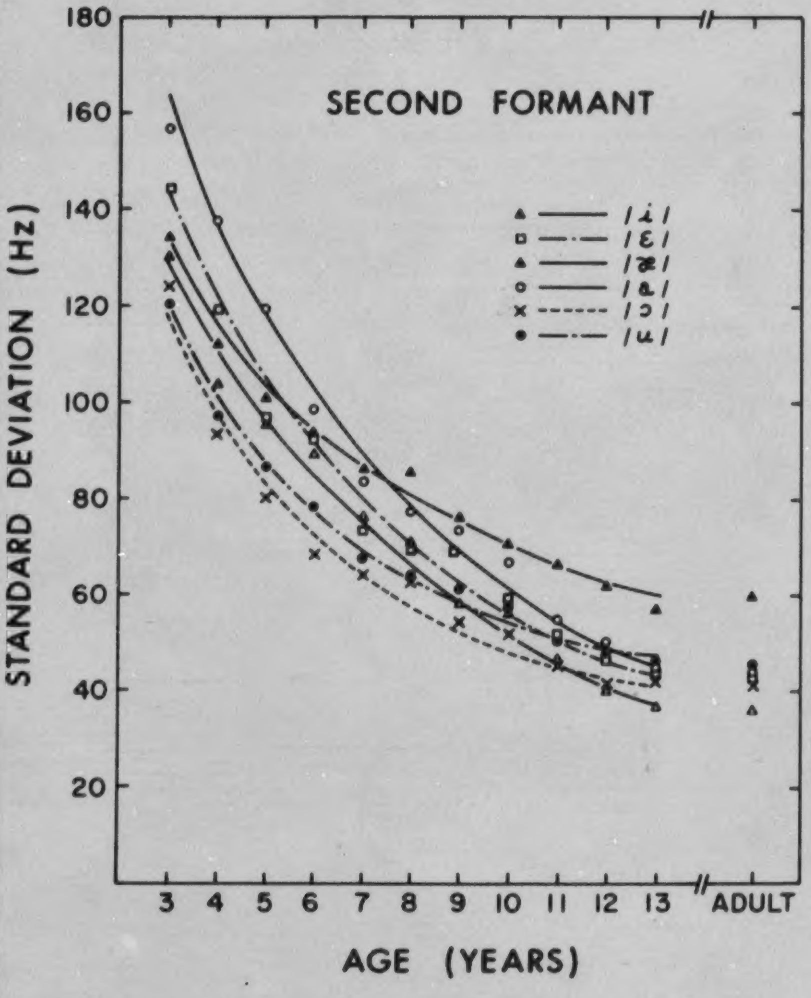

FIGURE 2 Intrasubject variability in formant 2 typical of the different age groups, as a function of age. Each point represents the square root of the average variance for each age group. (Reprinted with permission from Eguchi and Hirsh.[1])

explosion in the word "blue" and the onset of phonation corresponding to the letter "l"—which is about 40–50 msec. Such intervals constitute an integral part of speech, because their length helps the listener identify sounds. Here again we measured variability. If the child says "blue" five times in a sentence context, is the interval always 40–50 msec, or does it vary?

Figure 3 shows that variability changes with age. The mean interval between the "b" and the "l" or the "p" and the "e" in "pen" does not change over time, but the average intrasubject variability around that mean decreases sharply with age. It is interesting that it approaches its asymptotic minimum (corresponding to the adult value) at a much earlier age than do the spectral features of the vowels. Precision in the temporal feature, at least in English, is extremely conspicuous. Whether this

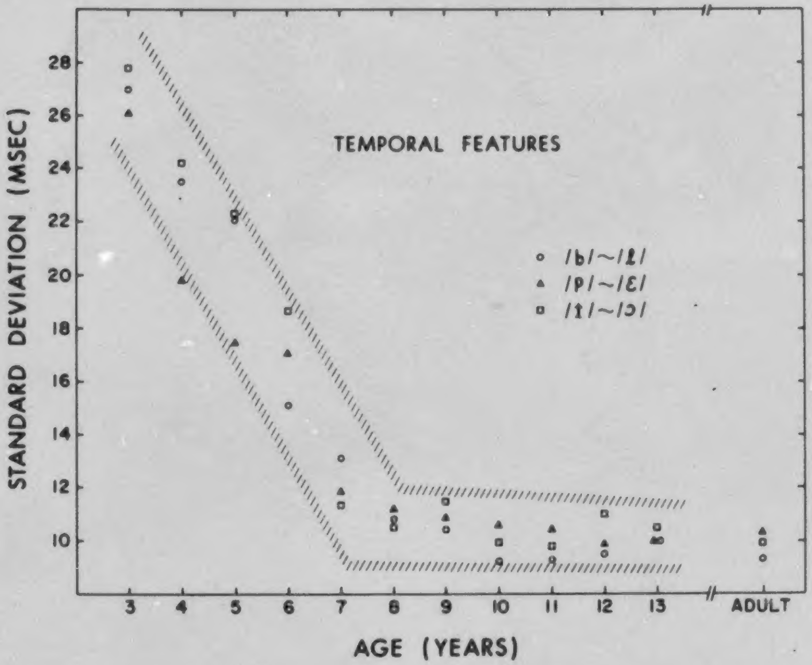

FIGURE 3 Intrasubject standard deviation of three temporal features of the words "blue," "pen," and "tall," as a function of age. (Reprinted with permission from Eguchi and Hirsh.[1])

is because temporal features are more important in language learning than the spectral features of vowel identification is difficult to say. There are some complications related to phonemic load over time that we cannot go into now.

RELATIONSHIP OF LANGUAGE COMPETENCE AND READING

Handicap of Deafness

Deaf children who have not learned to speak learn to read with great difficulty. It is interesting that they are taught to read by teachers who use paper and pencil to institute communication. Until then, there has been no communication between that child and others except by signs. If we could find the printed correlate of the conventional signs of the deaf, perhaps we could teach them. Instead, alphabetic teaching is used, but through the medium of interpersonal written communication. In the case of deaf children who learn to speak through use of what residual hearing they have, the teaching of reading is somewhat simpler, and it can begin at a much earlier age (about 3 or 4 years) to make up for what will be lost time. Even so, throughout the years of the elementary school, deaf children, even those who speak, generally maintain a retardation in reading of about two grade levels.

Let me give the essentials of a study done by Hartung[2] in connection with his recent Ph.D. dissertation at Washington University. Experiments have demonstrated that children can produce nonsense syllables (trigrams) when they are pronounceable more easily than when they are not pronounceable. For example, the syllable "mox" will be recognized correctly more often than the group of three letters, "mxo," which does not conform to normal spelling traditions in English and is very difficult to pronounce. Dr. Hartung wanted to study not only the variable of familiarity with the code to tie together the graphemes and phonemes, but also the variable of letter familiarity; therefore, he used trigrams that were made up of Greek letters.

Hartung asked his subjects to do two tasks. In the first, after a series of flashes lasting a couple of seconds, he asked children to tell whether a particular letter was present. No reproduction was needed. For three exposures, normal children about 8 years old correctly identified the presence or absence of a single Greek character in about 78% of the trials, and deaf children of about the same age, in 75%. We conclude there is no significant difference in identifying Greek letters. In identifying the letter "a," hearing children after brief exposures were correct in 89% of trials and deaf children, in 71%, a significant difference.

When trigram reproduction was required–writing down all three letters–the deaf children were at an even greater disadvantage. For example, after brief exposures of pronounceable trigrams, the deaf children were 31% correct in their reproductions and the hearing children, 71%. For the nonpronounceable trigrams, deaf children were only 15% correct and hearing children, 48%.

We expected the deaf children to do more poorly than the hearing children on trigrams in general. What was interesting was that they did even worse on the nonpronounceables than on the pronounceables. In short, in the process of learning what speech they had managed to learn up to the age of 8 or 9 years, they had acquired enough of the rules of correspondence between phonemes and graphemes for that difference to show up in their responses.

There was considerable spread in these data. In normally hearing children, there was no correlation between these flash-recognition scores and reading, as measured on a MacGinty-Gates Form C, but in the deaf children, there was a significant correlation of 0.5.

I am not going so far as to say that we read with our ears, although I recognize that different specialists view things from their own points of view. George Sperling has said that short-term memory very often involves a conversion to auditory storage. I suggest that such storage is especially important when part of the information being stored is of a language–that is, is verbal. There is something special about verbal responses that I do not quite understand.

Stimulus–Response Compatibility

We were interested in information processing and so-called compatibility between response and stimulus. We have tried to investigate responses to verbal and nonverbal stimuli in two modalities, hearing and vision. We restricted ourselves to a vocabulary in which the objects were named by relatively simple words, like "bell," "cat," "dog," and "baby" and were sound-producers themselves. We could flash pictures of objects and of the printed words for them and play the spoken words and the sounds of the objects (the bark of a dog, the meow of a cat, the cry of a baby, the sound of a bell, and so on) on tapes. This gave four different vocabularies, two visual and two auditory; considered the other way, two were verbal and two were nonverbal.

Reaction time was measured for three different responses. The first was the pressing of a key on which a picture of the object appeared, and the second was a similar response, but with a printed word on the key. The third response was merely a spoken identification or verbal response. One hypothesis from the notion of stimulus–response compatibility was that the reaction time would not increase with number of alternatives for those combinations of auditory-verbal stimuli and spoken responses, visual-picture stimuli and visual-picture keys, or visual-verbal stimuli and visual-word keys. Other combinations would show less compatibility between the form of stimulus and the form of response.

The subjects were 30 women, 19–24 years old. Our first experiment was a two-alternative choice. There were two keys, one with the word "dog" and the other with the word "bell"; the subject was to push the appropriate one when she saw or heard a stimulus. As we increased the number of choices to eight, there were four keys in each of two little semicircles, so that the eight fingers could rest on them. We even controlled for finger preference by using different arrangements of the keys for different subjects. The reaction time did increase with the number of alternatives, as predicted, when the response was pressing a key.

What was peculiar in this series of experiments was that, when we asked the subject simply to tell us what the visual word was, the verbal-response reaction time did not increase with the number of alternatives (which were told to the subject before the trial). Apparently, one does not have to translate modalities. This result appears to be independent of the stimuli and appears also to have to do more with a verbal response as such than with the relationship between the response and the stimulus.

The results were similar with an auditory stimulus. A word was spoken on a little Language Master card, and the subject was supposed to push the key that had the appropriate word or picture printed on it. The reaction times followed the general rule except for verbal responses, which did not increase with the number of alternatives. The general case appears to be that the reaction time increases with the number of stimulus alternatives (i.e., the slope of the line relating these two quantities is high), whereas in the verbal case, the reaction time stays relatively constant over different numbers of alternatives (i.e., the slope of the function is low or near zero).

The fourth stimulus was the auditory object, the sounds. They are probably not as easy to identify; it takes longer to comprehend what they are. Here, again, the slope of this function relates the verbal-response time to the number of alternatives and was much lower for verbal-response than for press-response time.

I am sure that, if these subjects practiced over and over again for many months with these press responses, we could bring the regression slopes down. Several workers have already shown that, if one practices a connection between a response and a stimulus mode for a long time, one experiences no increase in reaction time with number of alternatives.

The point of these data is to show that long-practiced correspondence is already present by virtue of "being part of the language." The stimulus mode—whether it is auditory or visual, verbal or nonverbal—does not make much difference. The press-response time increases with the number of alternatives, but the verbal- or vocal-response time does not.

LANGUAGE COMPETENCE AND SOUND

There are obviously contrasts between the reading of printed language and the understanding of speech. I should point out first that, although every known society communicates by talking, many societies are completely illiterate.

A second point is extremely important: sound can arrive at the ears from any direction. Images fall on the eye only from sources that one is looking at. Thus, those dealing with vision must be concerned about eye movements, fixation, and so on, to get the target to the macula. But the attention of which Dr. Hochberg has spoken takes place as sensory information goes into the auditory system, because we can hear from all around us; we do not need to be oriented toward the source. If we extrapolate to exposure to spoken language, as opposed to written language, there are hours of spoken language impinging on our ears all day long. This occurs particularly in the case of a growing child, whereas written or printed language becomes part of his stimulus input for relatively short periods.

A third point, mentioned earlier, is that the sound of speech is continuous, and the listener breaks it up into appropriate chunks, because he happens to be a member of the same language community as the speaker. In the case of reading, some of that chunking is done by punctuation and the spaces between words.

I do not know about the visual counterpart of this, but language modifies some kinds of nonlanguage auditory perception. The Haskins Laboratory work[3] has demonstrated that some kinds of discrimination of nonverbal stimuli are sharpened in the parts of the stimulus dimensions that correspond to the boundaries between different phoneme categories. As far as I am aware, no one has suggested that some aspects of visual perception are modified by the very process of reading. As a trivial example, do letter-like forms become more discriminable than non-letter-like forms after a person has been using letters for a while? My suspicion is that they do not. Another aspect of modification is segmentation. The way in which one listens to speech sounds is quite different from the way in which one listens to other sounds. The language rules that one has internalized in a sense control auditory "glances."

There are probably other kinds of auditory subsets that follow the same rule and illustrate the same principle. For example, Morse-code telegraphers group nonverbal auditory stimuli as we do when we listen to speech sounds and as careful listeners do when they listen to various kinds of musical passages.

Another kind of contrast has to do with critical age. It seems clear that, if a deaf child is identified before the age of 1 year, he can be prepared by suitable auditory stimulation to use his residual hearing better for the learning of speech at the age of 2 or 3 than if he does not start being stimulated until the age of 2 or 3. Although that is not quite a critical age for learning spoken language, it is something like "if you don't catch it this early, then it isn't going to be as good for general auditory reception later." We do not know the critical age for learning speech, but teachers of normal speech development have suggested that, because some stages of syntactic development are characteristic of the normal child at the age of 1 or 2 years, this is the age at which speech learning must begin.

We have been told about similar observations on the visual system in handicapped children of one sort or another. For the visual system in general, I would be extremely interested to know whether a deprivation of the printed word has any serious consequences for learning to read. Or can we just as well start at the age of 10 without suffering difficulty?

SENSORY DEPRIVATION

I have not much mentioned deaf children, who afford, perhaps, a natural experiment on the question of the effects of sensory deprivation. I suppose that we can think of the congenitally deaf child as being like the congenital cataract patient–not in terms of the underlying pathology, but in the sense that we can, at some time after birth, alleviate the deficiency. In the case of a blind child, we remove the cataract; in the case of a deaf child, we amplify everything by about 60 or 70 db, and the effect is roughly comparable. He will not hear everything, to be sure, but all deaf children of my acquaintance have some sensitivity to frequencies up to about 500 Hz. I will not discuss whether the sensitivity is auditory or tactile; both can be used as information receivers, and both seem to benefit from early stimulation.

The deaf child who is left unattended until the age of 6 or 7 years can be taught to speak only with great care and difficulty. When sound was amplified sufficiently so that deaf children would respond, R. Gengel (in a doctoral study now being completed) found poor discrimination as a result of auditory sensory deprivation. An example is the child who can hear tones at 110 db but cannot distinguish frequency in the low-frequency range. A trained person with normal hearing can distinguish a frequency of 500 Hz from one of 505 Hz. These deaf youngsters on the average heeded differences of around 80–100 Hz; that is, they could not discriminate unless the frequencies were first 500 Hz and then jumped up to about 600 Hz. After about 3 months of training, the poor discrimination almost disappeared. He never got them down to a 5-Hz difference, but they did get down to 10 or 12 Hz, which is the difference that an untrained observer could probably distinguish.

We do not have more information like this for the ear because we do not have the elegant battery of clinical tests that we have for visual function. We measure the sensitivity by making an audiogram, and often that is the sole basis of information on what a child can hear. There are dozens of tests for visual function, but we do not have their analogs for auditory function. Ordinarily, we use lists of words and ask the subjects to discriminate them. If a child is 2 or 3 years old and has not spoken yet, the testing routine is difficult.

I have one suggestion for those who must face this problem of the long-deprived child, such as an underprivileged child from the ghetto who is very retarded in reading: to get him to read better, get him to talk better.

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DISCUSSION

MR. ADAMS: When a person is listening to a spoken message, the temporal order of the arrival of the message is in the control of the speaker, not the listener. When a person is reading a written message, the temporal order of the arrival of the message is in the control of the reader, not the writer. Those facts are due to the different properties of the space (or medium) through which messages are transmitted. When a message is transmitted through acoustic space, it occurs in real time and exists only in the temporal dimension. The decoding of acoustic messages obeys special rules associated with acoustic space. When a message is transmitted in two-dimensional visual space, the message is "stored" in a medium that has no temporal dimension. Written language is acoustic language encoded in visual-form space; a written message has no temporal dimension but only the two dimensions of length and breadth. It is the responsibility of a reader to supply the temporal dimension according to the rules of the written language that govern the direction of the visual scanning process. If a reader scans the letters "d o g" from left to right, he will decode the message to read "dog." But if he scans them from right to left, he will decode the message to read "god." I suggest that this directional scanning may account for the reversals that we sometimes see with a retarded reader.

So we have come full circle: visual chunking is not done for us by the page; a reader must supply the chunking in the same way that he supplies the temporal chunking when he listens. One of the big differences between decoding language stored in visual space and decoding language stored in acoustic space is that a receiver decoding language stored in visual space must know the rules for supplying the temporal dimension. But the responsibility is relieved for him when he is decoding the message in acoustic space, and many of our problems in the strategy of teaching reading are due to overlooking this crucial fact.

I think this problem was identified by one of the experiments in Project Literacy conducted at Cornell. It was discovered that the child must learn the rules governing the visual cues associated with recognizing the visual boundaries of words. Knowing these rules is crucial for successfully learning how to decode written language. I do not recall all the details associated with that particular experiment, but it will suffice to say here that members of the Project Literacy staff reviewed the teacher's manuals that accompany commercially prepared children's readers, such as the basal reading series prepared by children's textbook publishers. They were looking for specific instructions to the teacher on how to prepare specific lessons that overtly instructed a child on matters pertaining to the visual boundaries of words. After the review, the Project Literacy staff surmised that teachers are neglecting this important aspect of learning to read. Indeed, at no time did the teacher's manuals mention that not only alphabet recognition must be taught, but also word recognition and paying attention to both the white spaces between letters and the white spaces between words, sentences, and paragraphs, because they contain important visual cues to the decoding process. A child must learn these rules in order to make correct decoding decisions, because there are a finite number of letters that can be grouped in infinite ways to make up infinite words, which can be combined in infinite ways to make up infinite messages.

I just wanted to take issue with you in a friendly way and ask how you think the chunking is being done by ghetto children when they decode messages in the visual mode, as opposed to the acoustic mode.

DR. HIRSH: My main emphasis should have been restricted to the identification of words. I am less concerned here with temporal order than with segmentation. Even though children may not yet know the significance of the rules that tell them what to do with the larger white spaces between the words, those white spaces do appear in the visual stimulus pattern. No such spaces appear in the auditory message. If I showed you an oscilloscopic tracing of the waveform of the sounds that make up a sentence, you would see that there are few if any silences and that the acoustic message is essentially a continuous sound whose internal structures change with time. One must have learned to speak and to listen in a particular language in order to organize pieces of that continuous sound in such a way that they will correspond with the morphemic elements of the language. Such separation or segmentation must be put in by a listener as he listens. Similar segmentation may also be a part of the reading process, but at least the segments are more clearly marked with spaces on the printed page.

DR. BOYNTON: I think Dr. Liberman of the Haskins Laboratory at the University of Connecticut recently suggested that the basic processing mechanisms required for the appreciation of speech sounds (which vary from one language to another, but perhaps not very much) might be built in as part of the sensory regulating apparatus. I do not think that anyone would suggest that such equipment could conceivably be built in for the processing of the visual counterpart of letters and words. This would constitute a fundamental difference between reception in the two modalities.

DR. HIRSH: If what he has said is as general as saying that humans contain a predisposition for spoken language, then I agree. If he is implying that there are various built-in categories for auditory perception and phonemes, then I am not sure that I would go that far. These become built in very soon, I suggest, but they are certainly different from one language to another; and certainly no neurophysiologist, to my knowledge, has discovered a feature extractor that corresponds with phonemic features. They have found a feature extractor that corresponds with some interesting acoustic features, such as whether the tones glide upward or downward, something like edge detectors or angle detectors if you like, but none that is specifically phonemic.

EARLY EXPERIENCE AND LEARNING IN VISUAL INFORMATION PROCESSING

AUSTIN H. RIESEN

Effects of Visual Environment on the Retina

The study of transneuronal effects on neural microanatomy has a rather long history, which I will not attempt to repeat here. (It has been brought up to about 2 years ago elsewhere.[9]) The forward march in this area has been increasing in momentum. The phenomenon of transneuronal degeneration is no longer just a curiosity, as it was regarded by many in the 1940's, particularly after LeGros Clark called attention to it in his study of the lateral geniculate nucleus of primates. Transneuronal effects are turning out to be very extensive, and much more needs to be learned about their time course. They are clearly both upstream and downstream effects within the nervous system. The effects are not limited to early development, although they are considerably faster during infancy. Later, some are rather slow.

EFFECTS OF LIGHT DEPRIVATION ON PROTEINS

One of the more dramatic landmarks dealing specifically with changes in the retina was a study by Brattgaard.[2] He reared rabbits in darkness to the age of 10 weeks, and showed that the retinal ganglion cells were markedly retarded in development. He found that 3 weeks of light stimulation after 10 weeks in the dark brought about a recovery of some cells but virtually no recovery of others. There was a great increase in variability of RNA content and also in the relatively stable protein content of the cell body, both nucleolar and cytoplasmic. At about the same time, we were rearing chimpanzees in the dark; we found that, if we kept them in the dark too long, the ganglion cells not only lost out in the race for protein, but died and disappeared. The first signs of this effect could be seen by the age of 3 months as a disk pallor of the retina. The leads from Brattgaard's study induced us to look at RNA in dark-reared rats and kittens and in the remaining cells of the chimpanzees' retinal ganglia.

We found that impaired protein metabolism is common to the retinal cells of dark-reared rabbits, rats, cats, and chimpanzees. Rates of change from normal RNA levels appear to vary with species, when average values are examined, and with individual cells (as determined by measures of variability within a particular class of retinal cell). Special staining techniques make these cytochemical determinations possible. Using hematoxylin and eosin staining permitted us to determine only that large numbers of ganglion cells in chimpanzees and monkeys eventually disappeared[3] and that in cats the mean thickness of the inner plexiform layer of the retina was significantly reduced.[7]

At 90 days of age, normally reared rats showed more than seven times the concentration of RNA found in dark-reared littermates. The values for retinal ganglion cells were intermediate when the animals were reared in the dark for 90 days and then in normal diurnal lighting conditions for 60 days. We have not determined visual acuity in these animals, but they can discriminate on visual cliff and visual placing tasks.

In cats from 3½ months to over 3 years old, the differences between light- and dark-reared animals in cells of receptor, bipolar, and ganglion layers are similar in amount, but in no instance are they proportionately as great as those cited for the rat. By making photometric estimates of azure-B binding, we found that the cells from dark-reared cats had cytoplasmic RNA concentrations between 40% and 55% of those in normal cats, and animals given 1 hr of light daily had intermediate levels, about 60–75%. These data do not reflect additional observations that the mean cytoplasmic cross-sectional areas were also significantly reduced in cells of the dark-reared animals; that finding augments the differences in total cytoplasmic RNA.

Dark-reared chimpanzees and monkeys and one chimpanzee that was given normal light stimulation from birth to the age of 8 months and then reared in total darkness until the age of 2 years revealed markedly reduced cytoplasmic RNA concentrations in surviving retinal ganglion cells. Depending on the age reached before the primate was placed in the darkroom, the reduction in number of ganglion cells varied from zero to over 90%. When cell loss occurred, visual functions became impaired,[3,8] but reduced RNA concentrations were not necessarily correlated with losses in visual capacities. The late onset of deprivation, after up to 9 months of normal stimulation, produced more visual impairment in higher primates than in the other mammals that we have studied.

Figure 1 permits comparison of the parafoveal retina of a control chimpanzee with that of a chimpanzee that was reared normally to the age of 8 months, then lived in total darkness until the age of 24 months, and then lived in normally lighted environments until enucleation at the age of 8 years. The darker appearance of the retina on the right is due to its having been cut in a thicker section (15 μ) than the other (8 μ). More than 90% of the ganglion cells had disappeared in this animal and also in a second chimpanzee reared with 5 min of daily exposure to light from birth to the age of 33 months. These effects were found to be irreversible, whereas shorter intervals of total darkness in other primate subjects resulted in temporary pallor of the optic disk.

EFFECTS ON ELECTRICAL ACTIVITY

Loss of RNA and cell-volume reduction do not culminate in death of cells in the cat retina. The usual population of ganglion cells is markedly lower in cat, rat, or rabbit than in primates. This may permit the spontaneous firing of receptor cells, which occurs even in total darkness, to activate ganglion cells frequently enough to ensure their continuing viability. Converging pathways through bipolar fibers would lead to relatively greater use of the individual optic nerve fibers in cats, for example, than in monkeys, apes, or man.

Figure 2 shows electroretinograms (ERG) of a retina that had light deprivation. This represents a monkey that was normally reared and then put in the dark for a relatively short period, about 1 month. The b-wave, the upper deflection (as opposed to the a-wave, represented by

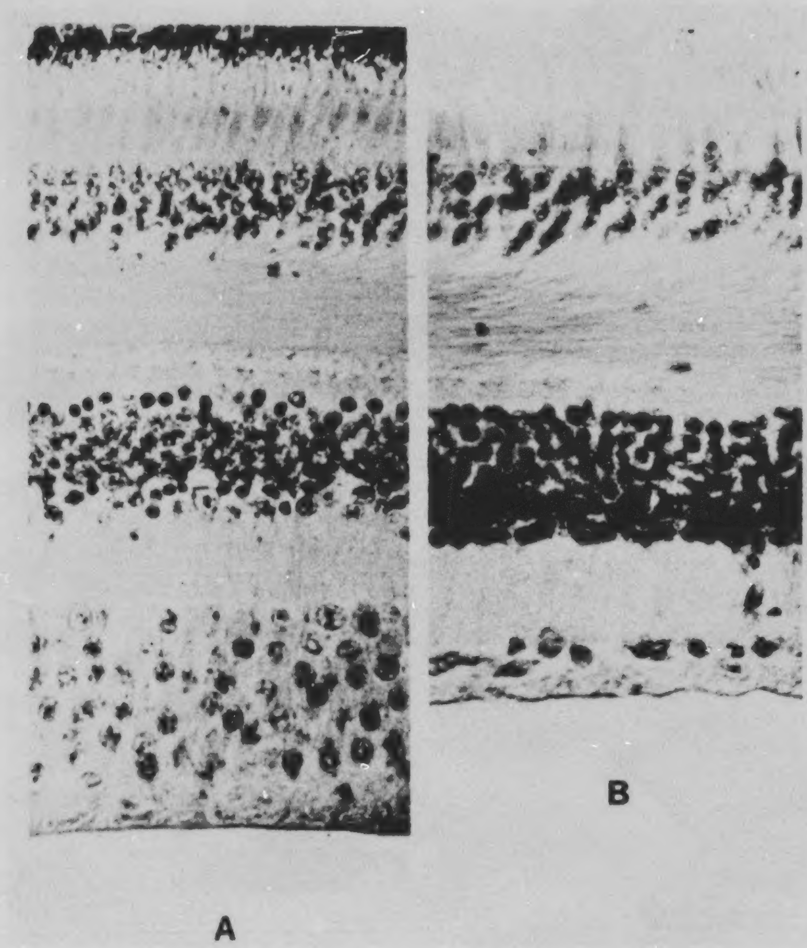

FIGURE 1 A, normal layers from a 26-month-old chimpanzee. B, Prolonged total light deprivation from the age of 8 months to 24 months followed by normal light to the age of 8 years results in marked loss of ganglion cells (bottom layer). (For full details of rearing and visual tests on these and other chimpanzee subjects, see Chow *et al.*[3] and Riesen.[8])

the first downward deflection), is, on the second flash, markedly reduced. The same phenomenon occurs in a kitten or an adult cat after as little as 1 week in darkness,[4] although its extent may be somewhat less. The lower traces are of later responses to flashes given at 2-sec intervals and show that these effects persist. Even when the flashes are spaced rather widely—as long as 10 sec apart—the recovery of the b-wave is still incomplete.[1]

Figure 3 compares some b-wave mean amplitudes in monkeys. The upper curve shows the reduction in the normal monkey's response to the second and third flashes. After 5 weeks in darkness, the b-wave responses of this group of animals to the second and third flashes, on the average, dropped to only one fifth or less of normal.

I will not speculate on the mechanism involved, but we do know that lack of stimulation in an adult or mature monkey, as well as in a mature cat, does something to retinal function, as measured by b-wave amplitude and its recovery.

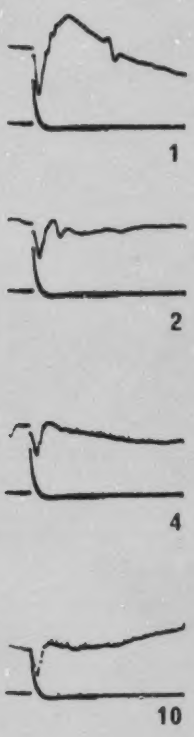

FIGURE 2 Individual monkey ERG records from surface electrodes, showing marked reduction of b-waves in responses after the first in a series of flashes. (Data from R. L. Ramsey.)

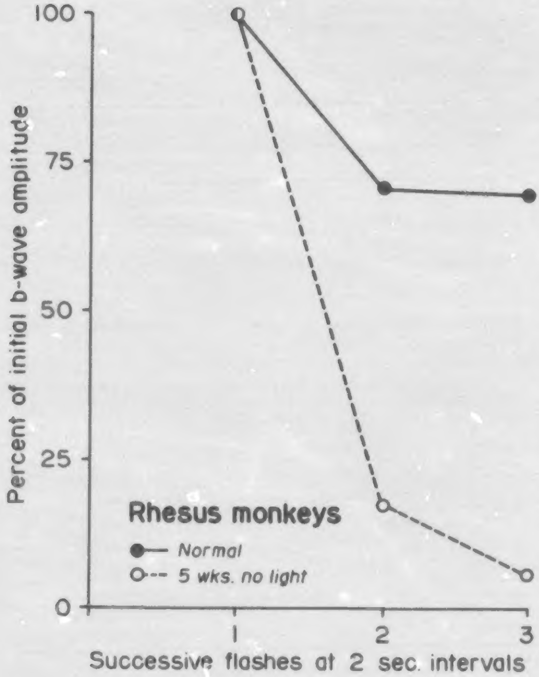

FIGURE 3 Amplitudes of ERG (b-wave) responses in normal and light-deprived monkeys.

BEHAVIORAL CORRELATES

I would like to indicate some behavioral correlates of lack of visual stimulation in the monkey at birth and shortly thereafter. The data (Figure 4) are from some acuity measurements that Dr. Paul D. Wilson made in our laboratory in Chicago. He raised animals in diffuse light of fluctuating intensity, rather than patterned light; the diffuse light was provided for 2.5 hr each day. On day 20 in this experiment, the animals were first brought into patterned light. Their visual acuity was measured with an optokinetic drum that had stripes of various widths to "pull" the eyes. There was a high degree of variability, but in the first set of three columns (representing three animals), the visual angle that was required to induce eye movements ranged from approximately 20 min to about 170 min on the first day. The subjects improved rather dramatically on the second and third days of patterned light experience, and there was continuing improvement as these 20-day-old monkeys were reared in a normal environment up to the age of 41 days. The black bars represent normative data taken from a study by Ordy *et al.*[6] Their infant monkeys were tested from the second day after birth, and their improvement corresponds well according to this measure of visual acuity.

These results fit the data of Wiesel and Hubel,[5,11] showing that it takes patterned light, not diffuse light, to improve the responses of edge-detector units in the visual system. We do not think that this measure of acuity represents retinal improvement itself. Rather, it is a visual-system measure that relies, probably, on midbrain oculomotor organization.

Other kinds of behavioral indicators show that patterned light is a

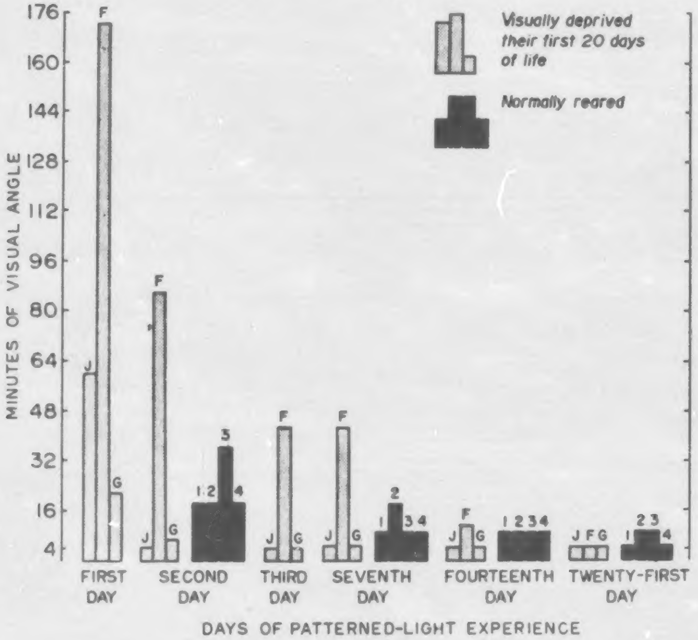

FIGURE 4 Improving visual resolving capacities of infant monkeys after 20 days of patterned-light deprivation (gray bars), compared with those of normally reared monkeys (black bars). The normally reared monkeys were studied by Ordy *et al.*[6] Data for the monkeys reared in diffuse light from Riesen *et al.*[10]

critical stimulating factor in the early days of visual development in the higher primates. Table 1 presents data from the rhesus monkeys used in Wilson's experiments.[12] The data on neonatally deprived subjects are ranges for 12 animals, of which six were raised in diffuse light for 20 days, and the other six for 60 days. The actual improvement in the particular behavioral measures listed did not differ significantly for the 60-day and 20-day groups, and they paralleled light-reared normal subjects that were studied from birth. Some of the differences indicate an advantage for the animals reared in diffuse light. Ocular pursuit of a moving light shows this slight advantage, perhaps: 1–3 days versus 3–12 days of patterned light experience. Inaccurate pursuit of objects is the exception, for this item first appears on days 6–13 in the 20-day-old or 60-day-old pattern-deprived animals, and on days 3–10 in the light-reared subjects.

For binocular convergence movements, we used a variety of stimuli for getting the best fixation response possible from a little point of light or a colored object, and then moved the stimulus toward the infant's face. The animals that had matured for 20 days under the diffuse-light conditions did markedly better. They showed convergence for the first time in the second week (days 7–11) after patterned light was initiated. The others did not show this behavior until after the third week (after birth). Accurate reaching to an object starts at about 2 weeks of age in both groups. Starting at 20 days or 60 days provides some advantages

TABLE 1 Age in Days at Initial Appearance of Visual Behaviors in Monkeys[a]

Visual Behavior	12 Neonatally Deprived Subjects[b]	Light-Reared Subjects
Consensual pupillary response	1–2[c]	3–7[c]
Ocular pursuit of light	1–3	3–12
Ocular pursuit of objects	4–12	4–28
Visual placing response	5–16	6–21
Inaccurate reach to object	6–13	3–10
Binocular convergence	7–11	21–35
Accurate reach to object	12–29	14–56
Visual cliff avoidance	11–34	–

[a] Derived from Wilson and Riesen.[12]

[b] Patterned light given 2.5 hr daily to neonatally deprived group, beginning on 21st or 61st day after birth.

[c] Ranges represent days of age for the normally light-reared subjects and days of age minus 20 or 60 for the deprived subjects.

that reflect either the motor activity that they have had from birth or maturation, or both.

In concluding, I would like to stress one point. This kind of finding, that stimulation is necessary for full development, is not restricted to vision and the retina. There are studies in other sense modalities, and we should take the data as a whole. We have to consider that development, as measured by various growth indicators and by anatomic and electrophysiologic methods, is maximized if there is appropriate stimulation, but that stimulation can also be excessive. There is evidence that 18–24 hr of excessive stimulation will also impair protein content of cells, whether measured by the RNA precursor index, by dry weight, or by total-protein assays. The notion of an optimum is clearly implied in the data, but the determination of the optimum remains for future investigation.

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DISCUSSION

DR. DENENBERG: You indicated that, in the rat, 90 days of visual deprivation produces 10% or 15% of normal RNA. But I recall reports that animals raised in total darkness still do exceptionally well on discrimination-learning tasks.

DR. RIESEN: Rats do, up to some age (about 140 days) when they begin to show impairment. That points to the fact that RNA content as a measure does not correlate well with seeing, and I hold no brief for the measured quantities of RNA protein precursors as an index of whether these systems have been organized.

I should make one other point here. Once there is a visual system functioning well, one can impose conditions that will eventually result in a ganglion-cell depletion, probably even in ganglion-cell death. Our chimpanzees performed better visually about 2 years before we sacrificed them; then, their performance worsened up to the time of sacrifice, 4 years after they underwent early deprivation. The early deprivation can, indeed, leave a partially functioning system, and yet eventually lead to death of the system.

One animal, Snark, was seeing poorly; we are sure his visual acuity was low when he was 4 years old, and he was around 10 years old when we did the histologic examination, by which time he was hardly seeing at all. What the sections showed was a loss of ganglion cells. He also had pallor of the disk, indicating an unhealthy optic nerve, from the time when he was still seeing some things rather well. He was groping around finding things slowly, using what vision he still had, and he had pupillary responses that were still fairly lively.

We have data from tests of rather difficult form discrimination in the cat. We reared the animals in darkness for the first 5 months and then light for 5 months, or light for 5 months and then darkness for 5 months. We found that the latter order results in normally rapid, complex form-discrimination learning. We used a block "X" and a block "N" that leaned over. The animals that were reared in the light first learned both form and movement discrimination at 10 months of age as rapidly as any normal cat of the many that we have tested in that situation. There were three animals in that experimental setting, and their scores were in the upper part of the range found for about 12 normally reared cats (see pp. 117-147 in E. Stellar and J. M. Sprague, Eds. Progress in Physiological Psychology. Vol. I. New York: Academic Press, 1966).

DR. DOTY: In the chimpanzee experiments, did the degenerative process continue after the animal was restored to normal lighting conditions?

DR. RIESEN: Indeed it did–both in the animal reared in darkness from birth and the animal reared in the light from birth to 8 months and then in darkness for 24 months. The atrophy, as we can tell from ophthalmologic examination, not only did not improve in the light, but it gradually worsened. We could not tell how many ganglion cells were left in that intervening stage, but the restoration of the light did not save the system.

DR. VALVERDE: Did you mention that ganglion cells of the retina completely disappeared?

DR. RIESEN: In chimpanzees, 90% of the cells disappeared, and we have confirmed that with a couple of monkeys. In a shorter period, not so many of them disappeared, but there was a reduced count. This same loss of cells will occur in other, related nuclei. I am thinking particularly of lateral geniculate nucleus degeneration, described by Wiesel and Hubel, in the works that I cited. Of course, there have been many studies in which the actual end organ was removed; then, there was slow but progressive degeneration, until, after shrinking, some cells finally disappeared (see the paper in *Progress in Physiological Psychology*, cited above).

DR. LUDLAM: When you raise a diurnal animal, like a monkey, cat, or rabbit, in the dark, other things happen as well. Do these animals eat as well as the others? Are they exercised in the same way as the others? I can envision changes in protein caused by a lack of exercise, improper nutrition, and so forth.

DR. RIESEN: We frequently measured activity. Our chimpanzees totaled as much activity for a 24-hr period in the dark as in the light. The distribution of the activity was a little different: they tended to be highly active in the early hours of the morning. They were getting hungrier and hungrier, I suppose, instead of sleeping. They did not develop the usual sleep–wakefulness cycle, but they were as heavy as normally reared animals and they ate as much.

We handled, played with, and fed them in the dark. We learned quite early not to isolate these cats or monkeys or chimpanzees. Some animals that we used earlier in our work did not do very well, because we were not providing enough extra inducement for activity.

I do not think that this is a nutritional problem in any sense, although there were some indications that calcium metabolism was slightly affected: two of our chimpanzees showed somewhat later ossification-center appearance in the long bones of the body. We were studying various growth indices at the same time, and this is the only effect of that kind.

In the past we thought that the skulls of some were more brittle and thinner, but it was highly variable. Others that were dark-reared had normal skull thickness. We do not know what to say about that, except that, in some genetically highly variable animals like the domestic cat, some might require sunlight for utilization of vitamin D in calcification and others require only what we gave them in the food.

Many indices were more variable in the dark-reared animals, but enough of them were just right in all measures so that I am no longer very worried about the nutritional variable.

DR. ALPERN: I have the impression that there were changes in the electroretinograms and that histologically you found that the chimpanzees, in contrast with the cats, had particular kinds of ganglion cells. This is not characteristic of electroretinograms, in that in a clinical population you find perfectly normal ERG's in patients, for example, with extensive damage and degeneration in the ganglion-cell layers of the retina. I wonder how you would react to the following trivial explanation: If an animal is reared in darkness and has a vigorous pupil response to the first flash in the stimulus train—a response that kept the pupil contracted much longer than in control animals—one would expect that the reduced retinal illumination of successive flashes would evoke smaller ERG's.

DR. RIESEN: I do not know whether we have any data that would answer that. It is a good possibility. There were RNA losses in the bipolar cells and even in the receptor cell bodies in those eyes.

DR. LINDSLEY: You said that the flashes were spaced as much as 10 sec apart; it was not a train of repetitive flashes.

DR. RIESEN: No; it is possible to keep an animal's pupil down by giving it a vigorous flash of light, and it might stay down that long. Even your pupils, if you keep yourself in the dark for 30 min, would remain contracted that long. We (Science 134:1626-1627, 1961; Psychonomic Science 1:33-34, 1964) used homatropine to keep the pupil dilated, as did Cornwell and Sharpless (Vision Res. 8:389-401, 1968).

F. VALVERDE / A. RUIZ-MARCOS

The Effects of Sensory Deprivation on Dendritic Spines in the Visual Cortex of the Mouse: A Mathematical Model of Spine Distribution

INTRODUCTION

Dendritic spines, a sequence of postsynaptic structures, are small, thornlike projections on the dendrites of neurons in the mammalian cerebral cortex. They were first discovered by Cajal,[3] who believed that they represented normal morphologic formations, although many contemporaries repeatedly questioned their existence. Electron microscopy not only has demonstrated that dendritic spines are widespread in the central nervous system (as they are observed to be with the light microscope in Golgi-Cox and methylene blue preparations), but has shown that they are sites of synaptic contact with the same characteristics as those of synapses formed elsewhere with dendritic trunks.[8,17,18,25,50]

The distribution of fiber terminals in the dendritic pools of cortical neurons reveals some principles of common organization. Some of these principles may be deduced through the study of the distribution of the dendritic spines that represent an accurate imprint of the dendritic synaptic coverage. It has been found that the distribution of spines along apical dendrites reflects not only the functional maturation of the cells[2,10,11,34] but damage to them[14,15,44,45] and possibly the functional state of their afferent fibers.[43]

In a preliminary study,[47] we had found that the mean number of spines per consecutive segment along apical dendrites (the superficial ramifications being excluded) of the layer V pyramidal cells increases exponentially with distance from the cell body. These observations are based on the area striata of the mouse. Further analysis permitted us to partially adjust this exponential relationship in two groups of mice of the same age: controls and mice raised in darkness from birth up to the age of 24 days.[43]

Since the work of Mann[32] and Carlson[4] around the turn of the century, it has been known that significant modifications in retinal nerve cells resulted from prolonged variations in visual stimulation. Recently, Chow *et al.*,[5] Weiskrantz,[49] and Riesen[37] confirmed the existence of important alterations in the mammalian retina after prolonged light deprivation.

On the one hand, current studies have proved that modifications of sensory input and environmental conditions can affect the morphology of the cortex and some subcortical structures. Gyllensten[21] described statistically significant diminutions in the diameter of cell nuclei and in the quantity of internuclear material. He made these observations in the supragranular layers of the area striata in mice raised in the dark from birth to the age of 1 month. Wiesel and Hubel[51] found decreased mean cell areas in the lateral geniculate nuclei of kittens in which one eye was deprived of vision for 3 months. However, they reported that no obvious histologic changes were observed in the retinas, the optic nerves, the superior colliculi, or the visual cortex. Using the Golgi-Cox technique, Coleman and Riesen[6] have compared the dendritic fields of the stellate cells of the visual cortex in cats that were reared in the dark with those of their normal siblings. In dark-raised cats, they found shorter dendrites, a reduction in the number of dendrites per cell, and (using Sholl's procedure of concentric circles[4]) a reduction in the number of intersections of dendrites with circles centered around the cell body.

On the other hand, Rosenzweig *et al.*,[38] Bennett *et al.*,[1] and Diamond *et al.*[12] have demonstrated that an enriched environment positively increases the brain weight in rats. These authors have suggested that the changes might be due in part to further development of the dendrites. The suggestion was tested by Holloway,[27] who found convincing evidence that dendritic branching increases in rats raised in environmental complexity. Furthermore, Gyllensten *et al.*[23] have recently described hypertrophy of the supragranular layers of the auditory cortex after visual deprivation and have suggested that it might represent a compensatory mechanism.

From the foregoing accounts, it is evident that either a sensory deprivation or an increase in training can modify the fine structure of the central nervous system. These structural changes can be studied with some of our classical procedures for light microscopy. The purpose of the investigation to be described here was to verify whether the number of spines in the apical dendrites of the large pyramidal cells in the mouse area striata can be modified by decreasing the sensory information that reaches the visual cortex. It was found that the number of spines diminishes in mice enucleated on one side and in mice reared in complete darkness and that in both normal and visually deprived subjects the number of spines along apical dendrites increases exponentially with distance from the cell body.

We would like to describe some morphologic details of dendritic spines and their relevant afferent connections, the effects of unilateral enucleation on the number and distribution of dendritic spines, and a mathematical model that defines the distribution of spines in the apical dendrites of the layer V pyramidal cells of normal and dark-raised mice.

MATERIALS AND METHODS

The material for this study consisted of 153 brains stained by the Golgi method[48] from a closed colony of black mice derived from an inbred stock. They were collected over a period of 4 years from litters born after early spring matings.

The brains used were from animals in three groups: 73 animals raised under normal conditions up to the ages of 10, 14, 19, 21, 24, 36, 48, 78, and 180 days; 58 animals raised in complete darkness, as described elsewhere,[43] up to the same ages as the control group; and 22 animals unilaterally enucleated at birth and allowed to survive up to the ages of 24 and 48 days.

The spines were counted in about 1,200 complete apical dendrites of pyramidal cells of layer V in the area striata. The apical dendrites were arbitrarily divided into nine consecutive segments 50 μ long, and the spines on each segment were counted. The numbers of spines were plotted and punched on cards. Further processing for study and adjustment of the exponential relationship between the mean number of spines per segment and the distance from the cell body involved the use of IBM 7070 FORTRAN and Autocoder programs written especially for this work.

RESULTS

Connections of Apical Dendritic Spines in Visual Cortex

The synaptic terminals on dendritic spines can be observed easily on Golgi preparations. They appear in the form of short twigs derived from ascending or descending fibers that are closely parallel to the apical dendrites for considerable distances. These parallel fibers were identified as the descending principal axons or their collaterals of the superficial pyramidal cells, or the ascending or descending collaterals of stellate cells.[33,34,44] In other cases, a group of fibers approach the dendrite at various angles, leaving a number of clustered terminals over localized portions of the dendrite.[44] These two forms of afferent dendritic spine synapses (from parallel fibers and by crossing-over contacts) have been well established in many previous Golgi studies.

Figure 1 is a camera lucida drawing of a portion of an apical dendrite of a layer V pyramidal cell at the level of layer IV of the visual cortex. Dendritic spines are clearly visible as small short-side appendages throughout the entire portion of the dendrite. Several fibers (2–6) approach the dendrite to make synaptic contacts on the spines. Fiber 1 is the main descending axon of one superficial pyramidal cell. This axon partly follows the dendrite and appears visible in the lower part of the drawing, where it runs parallel to and contacts the dendritic spines (sp).

We have been particularly interested in the establishment of the circuit that relates the specific afferents of area striata with the apical dendrites of layer V pyramidal cells. On the one hand, the specific visual afferents might synapse directly on the dendrites. Several indirect arguments pointed this out in previous studies,[15,44] but, to our knowledge, this articulation has never been observed. The specific afferents appear difficult to stain with the Golgi method in subjects over 3 weeks old, and only in younger animals is the identification of such fibers clear; but the dendritic spines are not fully developed, and the synaptic formations involved in this connection may be absent. On the other hand, the specific visual afferents synapse on the dendrites of intracortical asso-

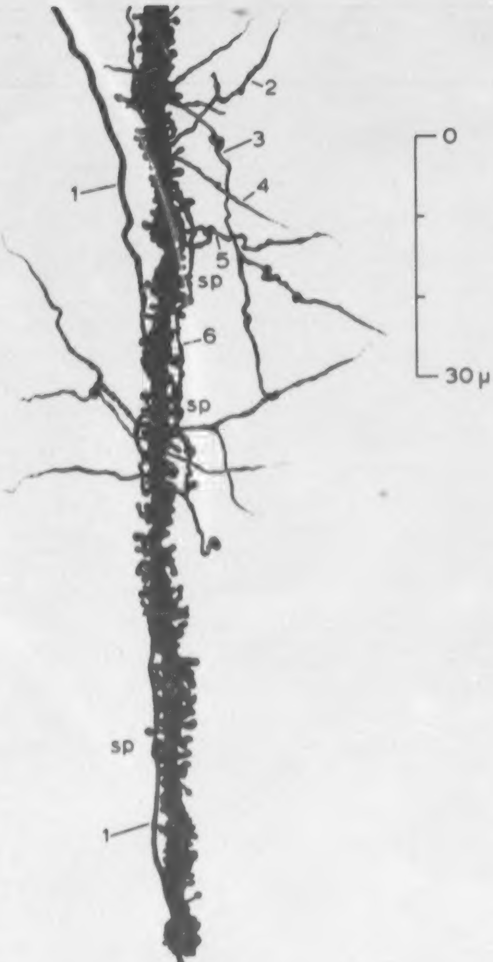
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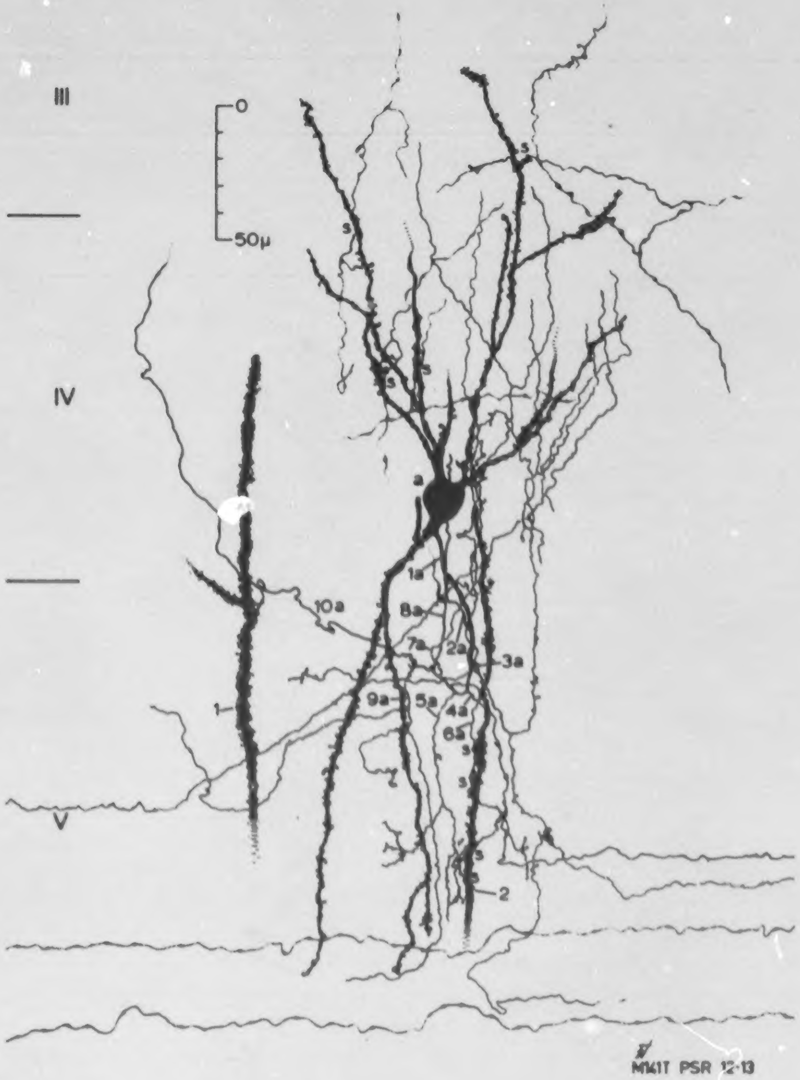
FIGURE 1 Camera lucida drawing of portion of apical dendrite of a layer V pyramidal cell of visual cortex of normal 48-day-old mouse. Golgi method.

ciation cells in layers IV and III, which in turn contact the apical dendrites through their short axons. We believe that this multisynaptic articulation is more frequent and most interesting, in that it increases the possibilities of modulatory effects on the pyramidal cells.

Figure 2 is a camera lucida drawing of one stellate cell in layer IV of the area striata of a normal mouse 48 days old. The dendrites radiate in two opposite bunches: the ascending one extends widely through layers IV and III, and a thick descending dendrite penetrates into layer V and bifurcates. The dendrites have numerous spines. The axon of this cell (1a) descends initially from its origin at the base of the descending dendrite and soon emits numerous collaterals (2a–9a). For example, collateral 4a originates in layer V and splits into a fiber that immediately turns horizontally and a branch that contacts the dendrite designated 2 at s. (The dendrite is a portion of the apical shaft of one pyramidal cell of layer V.) Likewise, collateral 6a emerges in layer V with several relatively large thickenings contacting the same dendrite 2 at two points s. Collateral 7a ascends for a long distance, traversing layers V, IV, and III; and collateral 9a descends in layer V after sending off a second collateral at a right angle (to the left in the figure). The main axonal fiber, now considerably thinner, pursues an obliquely ascending course (10a), giving off a small twig for dendrite 1, which is another apical shaft of one pyramidal cell of layer V.

Effects of Enucleation on Number of Dendritic Spines: Further Details of Axospinodendritic Contacts

In Golgi preparations, the apical dendrites of the pyramidal cells of layer V, which are 500–600 μ deep, as they ascend through layers V, IV, and III appear densely covered with spines, a series of short side appendages representing postsynaptic structures. The entire apical shaft, excluding the terminal superficial ramifications, can be arbitrarily divided into a number of consecutive segments (usually nine or ten in the mouse's visual cortex) each 50 μ long. Three of them were selected for counting dendritic spines: between 50 and 100 μ, between 150 and 200 μ, and between 250 and 300 μ from the cell body. The first segment is in layer V, the second in layer IV, and the third in layer III. The spines were counted in 540 selected segments in both areae striatae in mice enucleated of the right eye at birth, and the results were compared with those from normal mice of the same ages.


III
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MK11 PSR 12-13

FIGURE 2 Camera lucida drawing of stellate cell of layer IV of area striata of normal 48-day-old mouse. Golgi method. (Reprinted with permission from Valverde.[44])

In segments at the same distance from the cell body and from animals of the same age and condition, the numbers of spines per segment followed the normal distribution curve. Each segment contained a number of spines that remained, within statistically reasonable limits, around a mean value that was found to be specific for each distance from the cell body. The mean number of spines per segment varied, however, in animals of different age, diminishing significantly in segments in layer IV from the corresponding area striata of mice enucleated at birth.

Correspondingly, in the segment in layer IV from the affected areae striatae of enucleated animals, the diminution of the mean number of dendritic spines is statistically significant with respect to mean values from the same segment of normal animals. The difference is significant in both groups, 24 days old ($p < 0.001$) and 48 days old ($p < 0.005$). The difference is not significant when the comparison is between normal values and those obtained in the contralateral (right) area striata. For layer III, the same comparison reveals a difference significant beyond the 2.5% level. There was no difference in the number of spines in segments of layer V between enucleated and normal animals of the same age.

Figure 3 is a detailed camera lucida drawing of several portions of apical dendrites of deep pyramidal cells traversing layer IV. The drawing was made from the area striata of a normal mouse 48 days old. Figure 4 is a detailed camera lucida drawing of a field similar to that of Figure 3 but corresponding to the left area striata of a 48-day-old mouse enucleated at birth on the right side. Several portions of apical dendrites of deep pyramidal cells traversing layer IV clearly show the effects of enucleation. The number of spines has diminished considerably on dendrites 1–5. Dendrite 6 in Figure 4 is accompanied by a fiber coursing in the same direction, showing several short side appendages that contact the dendrite and spines. This fiber could not be identified, but it probably represents a descending axon or collateral of a superficial pyramidal cell, or an ascending fiber of a stellate cell. Axons of both types of cells have been observed frequently to lie parallel to and contact apical dendrites in this manner.

Figure 5A shows at the level of layer IV part of the apical dendrite of one pyramidal cell of layer V of the right (nonaffected) area striata from a mouse 24 days old, enucleated at birth on the right side. Figure 5B shows at the same level part of another apical dendrite from a pyramidal cell of layer V of the area striata from a normal mouse 48 days old.

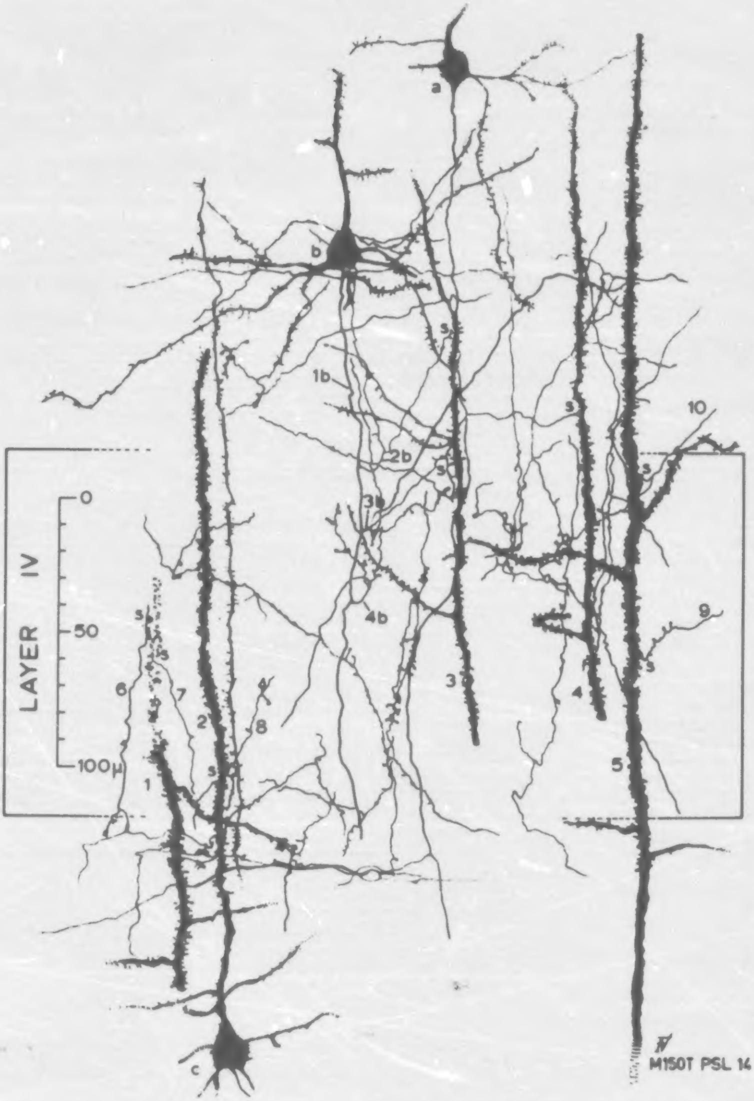
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FIGURE 3 Camera lucida drawing of portions of several apical dendrites of layer V pyramidal cells traversing layer IV of area striata of normal 48-day-old mouse. Details of synaptic contacts between various fibers and the dendrites can be seen. Golgi method. (Reprinted with permission from Valverde.[44])

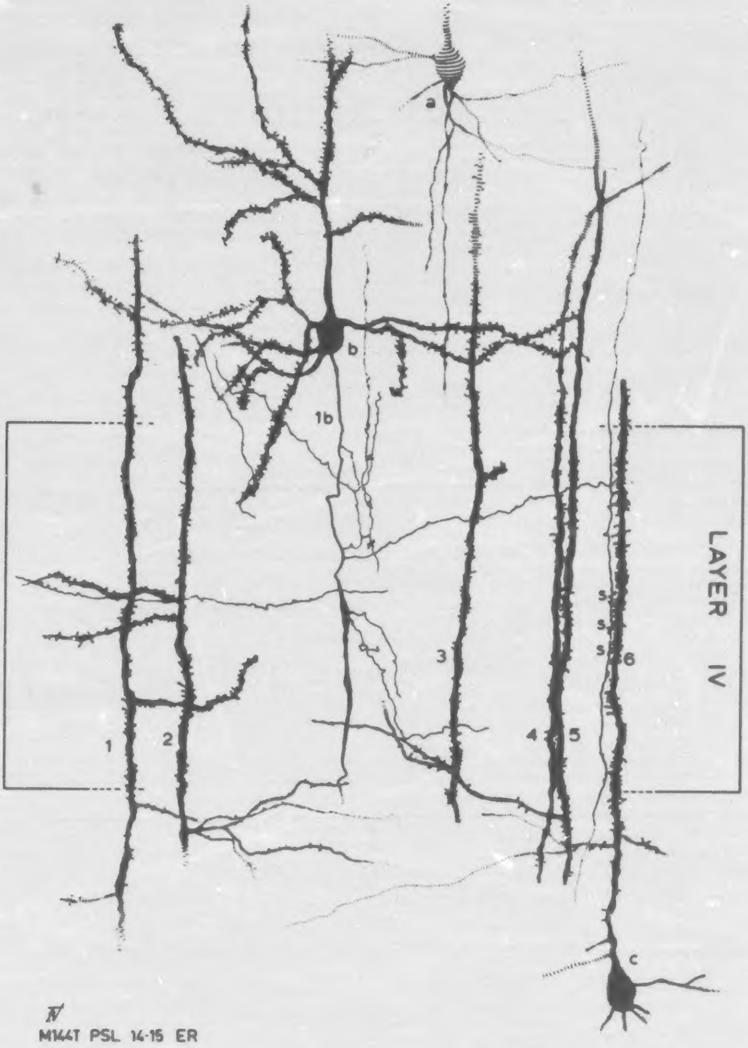
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FIGURE 4 Camera lucida drawing of portions of several apical dendrites of layer V pyramidal cells traversing layer IV of left area striata of 48-day-old mouse enucleated on right side at birth. Compare with Figure 3; note diminution of spines. Same magnification as in Figure 3. Golgi method. (Reprinted with permission from Valverde.[44])

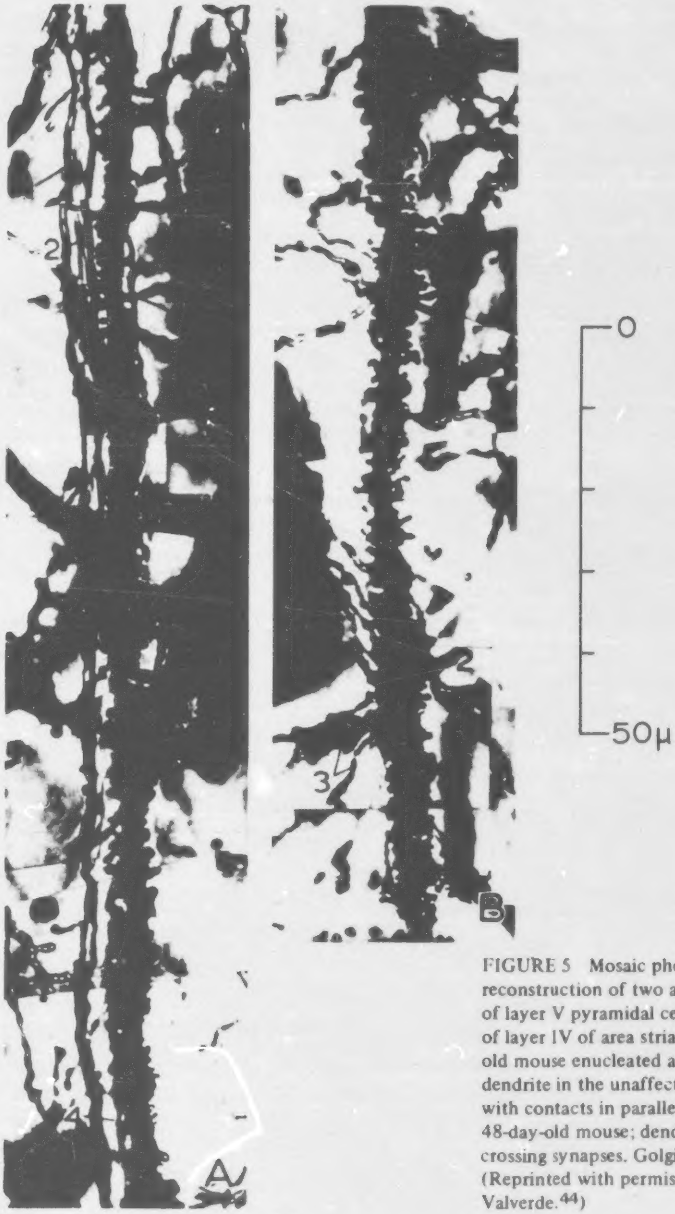

FIGURE 5 Mosaic photomicrographic reconstruction of two apical dendrites of layer V pyramidal cells at the level of layer IV of area striata. A, 24-day-old mouse enucleated at birth; normal dendrite in the unaffected area striata with contacts in parallel. B, normal 48-day-old mouse; dendrite with crossing synapses. Golgi method. (Reprinted with permission from Valverde.[44])

Dendritic spines are more numerous and thickly distributed in older animals. In Figure 5, afferent fibers cross the dendrite at various angles over a small segment, about 20 μ long, which appears densely covered by terminals of these fibers. It seems clear that contacts in parallel (Figure 5A) would have widespread effects over long dendritic portions, whereas crossing synapses (Figure 5B) would affect restricted parts of the dendrite.

Repeated observations showed that fibers synapsing in parallel on dendritic shafts of deep pyramidal cells represent either the main descending axon of a superficial pyramidal cell or its collaterals or ascending axonal branches of stellate cells. Synaptic contacts like those in Figure 5B have often been observed in layer IV. We believe that they represent terminations of specific afferent fibers over apical dendrites.

In Figure 6, the effects of enucleation at birth on the number of dendritic spines at the level of layer IV (affected area striata) in apicals of two layer V pyramidal cells (A, 24-day-old mouse; B, 48-day-old mouse) can be clearly observed.

Distribution of Apical Dendritic Spines in Visual Cortex of Mice Raised under Normal Conditions: A Mathematical Model

The number of dendritic spines was found to increase with distance from the cell body. We have previously defined this relationship for apical dendrites of pyramidal cells of layer V in the area striata of 24-day-old mice.[43,47] It can be expressed by the following exponential equation:

$$y_e = y_m(1 - Ke^{-Bx}), \tag{1}$$

in which

y_e = mean number of spines per 50-μ segment,
y_m = maximal number of spines on one segment,
e = basis of natural logarithms,
B = slope of corresponding regression line,
x = distance from cell body, and
K = value of $(y_m - y_e)/y_m$ when $x = 0$.

Equation 1 was found to define satisfactorily the distribution of spines along the first seven segments beginning in the pyramidal cell body, but it did not explain the decay of the number of spines occur-

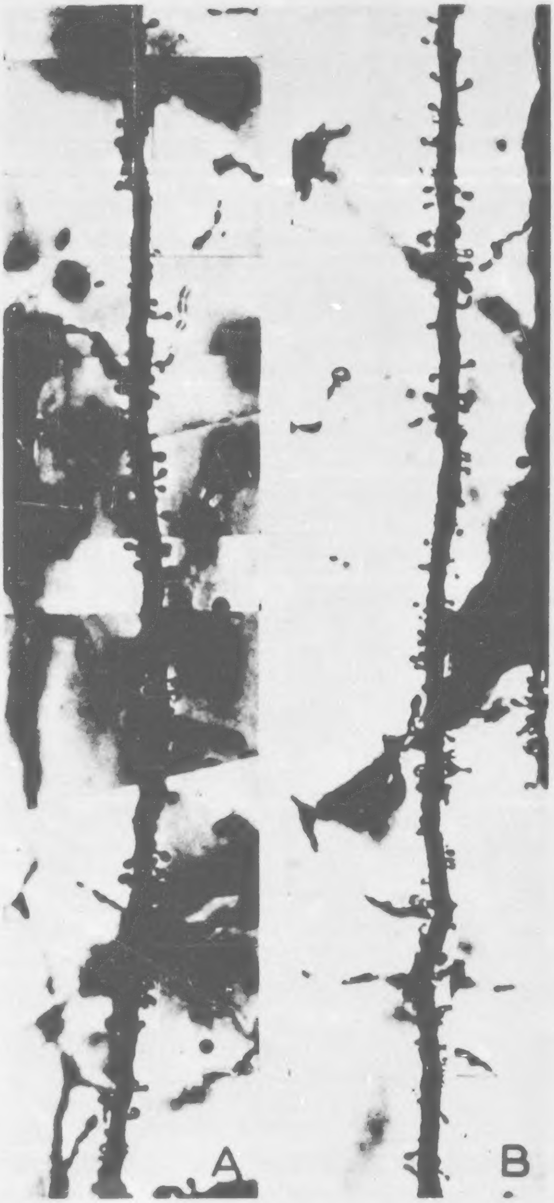

FIGURE 6 Mosaic photomicrographic reconstruction of two apical dendrites of layer V pyramidal cells at the level of layer IV of area striata. A, 24-day-old mouse enucleated at birth. B, 48-day-old mouse enucleated at birth. Both dendrites belonging to affected areae striatae clearly show diminution of spines. Compare with Figure 5. Same magnification as in Figure 5. Golgi method. (Reprinted with permission from Valverde.[44])

ring distally on the apical dendrite. We have studied the distribution of spines in mice 10, 14, 19, 21, 36, 48, and 180 days old raised under normal conditions and have obtained a sequence of different y_e's along the apical dendrites for each age. Figure 7 shows the observed distribution of the mean number of spines per segment in four representative age groups of normal mice. The mean number of spines for the first segment, between the body and 50 μ along the apical dendrite, corresponds to 25 on the abscissa; the mean number for the segment between 50 and 100 μ along the apical dendrite corresponds to 75 on the abscissa; and so on.

We then confirmed that y_e increases distally along the dendrite in all ages studied. But we observed, in the 24-day-old group[43] and in older

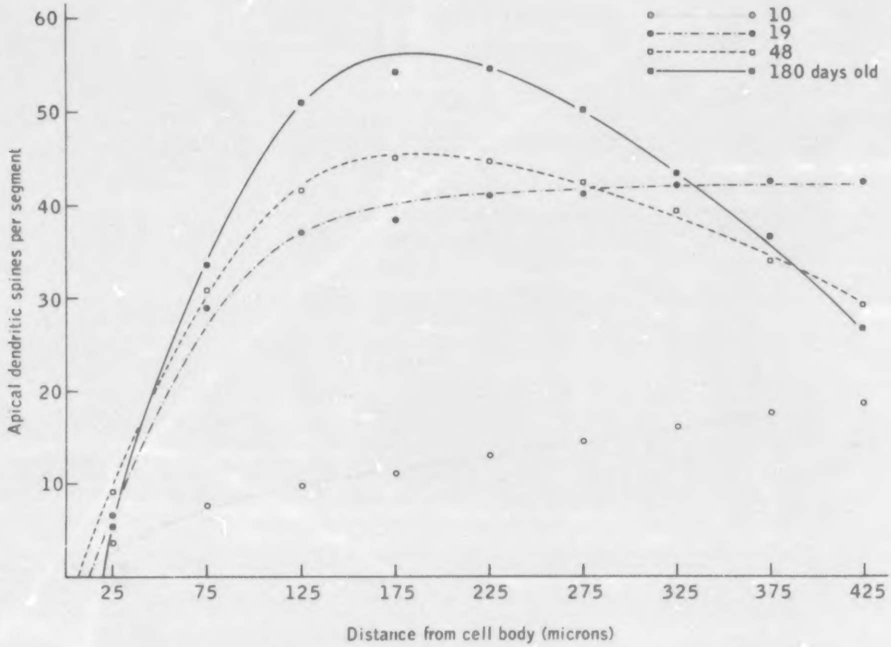

FIGURE 7 Number and distribution of spines in consecutive 50-μ segments along the apical dendrites of layer V pyramidal cells in the area striata. Sequence of mean values in four representative age groups of normal mice. Curves were fitted by hand. See text for details.

animals, that the number decreases gradually in more distal segments. The observation of these distributions suggested two factors: the potential factor B responsible for the increase in y_e distally along the dendrite and estimated by the slope of the regression line; and the inhibitory factor, IF, antagonistic with B, gradually hampering y_e in such a way that the combination might result in the distributions of Figure 7. It will be seen that IF becomes greater distally along the apical dendrite and also with the age of the animal, effecting the maximal decay of the mean number of dendritic spines in the last segments of 180-day-old mice.

Taking into account the specific properties of the exponential function, we have postulated that the observed distributions may be defined by this equation:

$$y_e = y_m \left(1 - Ke^{-Bx}\right) e^{-IFx}. \tag{2}$$

Thus, Equation 1 would be a particular case of Equation 2 when $IF = 0$.

To test whether Equation 2 defines the observed spine distribution, several programs were written for an IBM 7070. The data were fed into the computer on punched cards. The computer retrieved the mean number of spines (y_e) for each dendritic segment, the experimental distribution, and the 95% confidence limits on the basis of a Student's t-test. This program (RV-6801) was the first that gave the basis for further analysis of the spine distribution.

Through a specially programmed trial-and-error process (program RV-6803), the computer found the values of K, B, and IF that best fitted the experimental distribution by regression analysis.[46] After obtaining the values of K, B, and IF that gave the highest correlation, the computer calculated from Equation 2 the theoretical values of y_e and the chi-square values corresponding to the difference between the theoretical and experimental distributions, calculating the goodness of fit by comparing these chi-square values with a series of tabulated chi-square values stored previously.

The computer than printed out a graphic for each group of animals of the same age and condition with the experimental and theoretical distribution of the mean number of dendritic spines and their corresponding numeric values. The graphic output from this program, reproduced in Figure 8, corresponds to the distribution of spines along apical dendrites in normal 19-day-old mice.

With this program, we have studied routinely the distribution of

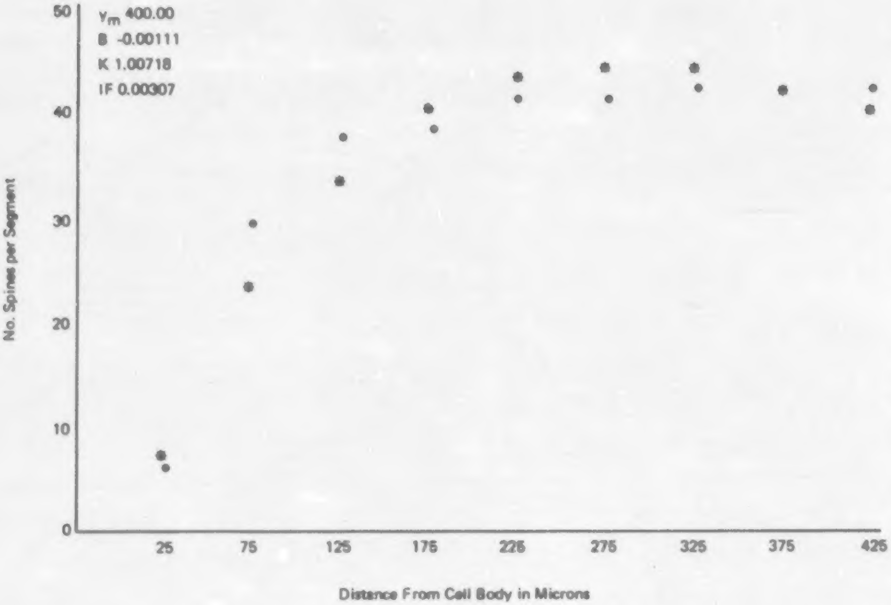

FIGURE 8 Experimental and theoretical distribution of dendritic spines along apical shafts of layer V pyramidal cells of area striata of four normal 19-day-old mice. Total dendrites, 49. Dot, experimental ordinate; asterisk, theoretical ordinate. When difference between theory and experiment is less than one spine per segment, only asterisk is shown. Data prepared from IBM 7070 program RV-6803. Chi value 2.409, 8 df, $p < 0.05$.

spines along apical dendrites of the layer V pyramidal cells of area striata in four groups of mice:

- Controls: 10, 14, 19, 21, 24, 36, 48, and 180 days old
- Raised in darkness since birth: 10, 14, 19, 21, 24, 36, 48, and 180 days old
- Enucleated on right side for study of distribution of dendritic spines in left area striata: 24 and 48 days old
- Enucleated on right side for study of distribution of dendritic spines in right area striata: 24 and 48 days old

In all four groups of striate apical dendrites, the computer obtained highly significant adjustments to the theoretical distribution formulated by Equation 2 with p values always less than 0.05 (< 0.01 and < 0.005 in several groups). Equation 2 is valid to describe the distribution of spines along apical dendrites in the pyramidal cells of layer V of the area striata of the mouse at the ages and conditions just mentioned, yielding specific values of the coefficients IF, B, and K for each age.

We have studied the relationships among the values of these coefficients and the age, T, of the animal, and obtained a series of graphics that suggest the existence of a relationship between IF and B and the age of the animal:

$$IF = IF_m \ (1 - IFK \cdot T^{-IFB}) \tag{3}$$

and

$$B = B_m \ (1 - BK \cdot T^{-BB}), \tag{4}$$

and also suggest a relationship modulated by an exponential function for the values of K:

$$K = K_m \ (1 - KK \cdot T^{-KB}) \, e^{P \cdot T}, \tag{5}$$

in which e = basis of natural logarithms, T = age of animal, and IF_m, IFK, IFB, B_m, BK, BB, K_m, KK, KB, and P = series of coefficients required for adjustments.

To test whether Equations 3–5 would satisfactorily represent the sequence of values, two programs (RV-6807 and RV-6808) were written for the IBM 7070. An adjustment better than $p < 0.05$ was obtained for all cases.

The system of Equations 2–5 constitutes a mathematical model that permits us to determine the distribution of the dendritic spines as a function of age. Through Equations 3–5 it is possible to calculate the values of IF, B, and K corresponding to a given T; then, by appropriate substitutions in Equation 2, the distribution in terms of number of spines per segment can be obtained for that animal. Program RV-6811 resolves the system of equations of the model and prints out a graphic with the spine distribution corresponding to a given age. Figure 9 is an example: after obtaining the data sheet of a predicted distribution corresponding to the 21-day-old mouse raised in darkness, we counted spines as usual through the microscope on a series of Golgi-stained brains and obtained a spine distribution that fitted the predicted distribution of Figure 9 ($p < 0.05$).

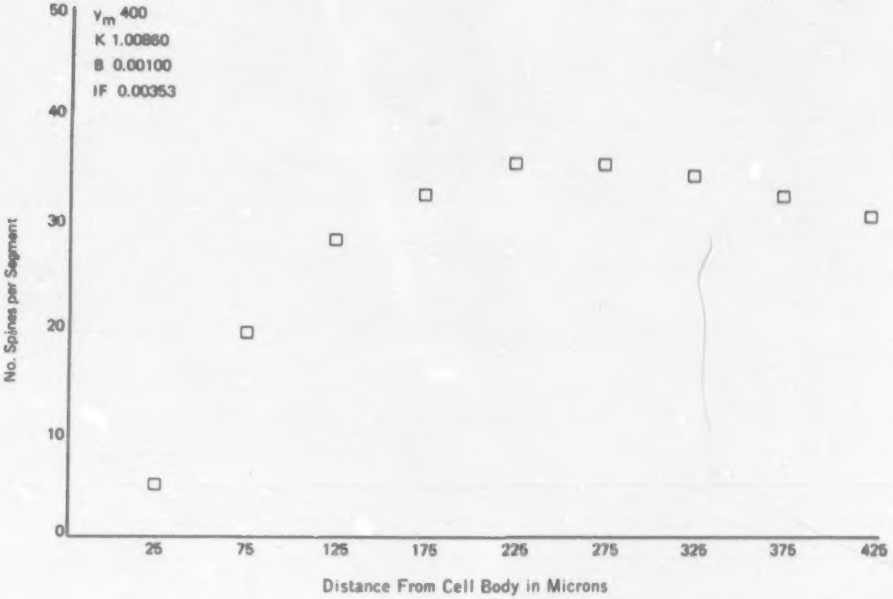

FIGURE 9 Predicted distribution of dendritic spines along apical shafts of layer V pyramidal cells of area striata corresponding to 21-day-old mice that were raised in darkness. These values fit ($p < 0.05$) the exper mental distribution obtained later from microscope countings. Data prepared from IBM 7070 program RV-6811.

Determination of Age on the Basis of Distribution of Dendritic Spines

With our mathematical model it is possible to solve the inverse problem; i.e., once we know the distribution of dendritic spines and the values of *IF*, *B*, and *K*, we can obtain the age of an animal according to the inverse of Equations 3–5.

We tried several animals to prove the validity of this process.[39] In some, we obtained *T* values differing by 15–20 days from the real ages of the subjects tested, but in others we got fairly good approximations of the true age. For example, the brain of mouse M240, whose age was not previously revealed to us, was stained by the Golgi method. We counted the spines on consecutive segments of 43 apical dendrites. The values for each apical dendrite were punched on individual cards and processed with program RV-6805 to obtain values of *IF*, *B*, and *K* corresponding to the minimal chi-square value. These were substituted in Equations 3–5 to obtain the corresponding T values. Identical T values in a given animal were never obtained. For mouse M240, 21 days old, we obtained a mean T value of 21.77 days.

Distribution of Dendritic Spines in Visual Cortex of Enucleated and Dark-Raised Mice

Mice enucleated at birth or raised in darkness are subject to a statistically significant diminution of the number of spines, which is most evident at layer IV in enucleated animals and throughout the apical dendrites in dark-raised animals of all the ages we have studied.

Figure 10 is a graphic output from program RV-6803 corresponding to the distribution of dendritic spines in the affected area striata (con-

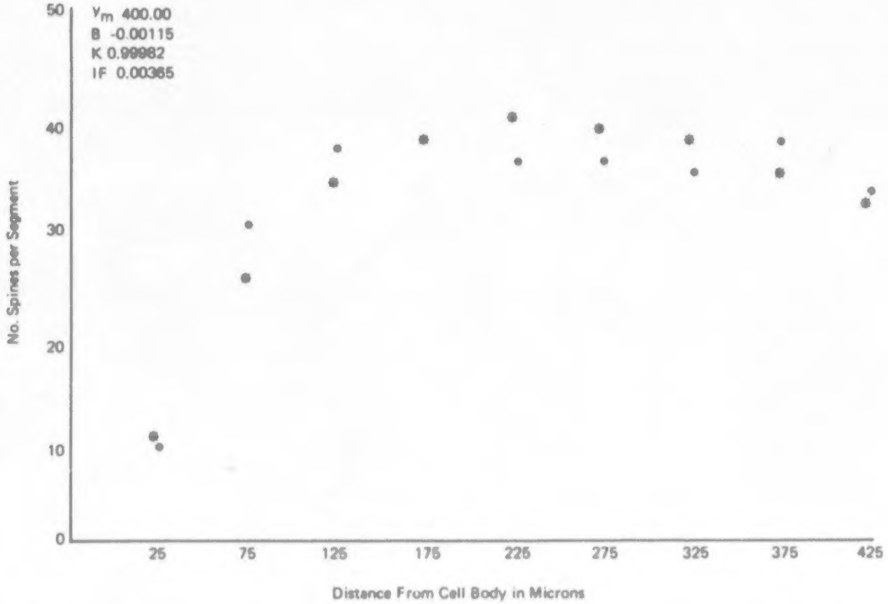

FIGURE 10 Experimental and theoretical distribution of dendritic spines along apical shafts of layer V pyramidal cells of affected area striata of four 48-day-old mice enucleated at birth. Total dendrites, 31. See caption of Figure 8. Data prepared from IBM 7070 program RV-6803. Chi value 2.257, 8 df, $p < 0.05$.

tralateral to the enucleated side) of 48-day-old mice enucleated at birth. The distribution can be compared with that obtained for normal mice of the same age (Figure 7). In enucleated mice, we observed somewhat lower mean numbers of spines per segment at classes 125, 175, 225, and 275 corresponding to the level of the apical dendrites traversing layer IV; the differences from the averaged numbers in normal mice were significant.

Figure 11 shows the graphic output from the same program corresponding to the distribution of dendritic spines in 48-day-old mice raised in darkness. In all age groups of mice raised in darkness that we have studied, highly significant agreements with the theoretical distribution formulated by Equation 2 have been obtained, with p always less than 0.05. The differences from the averaged numbers of spines ob-

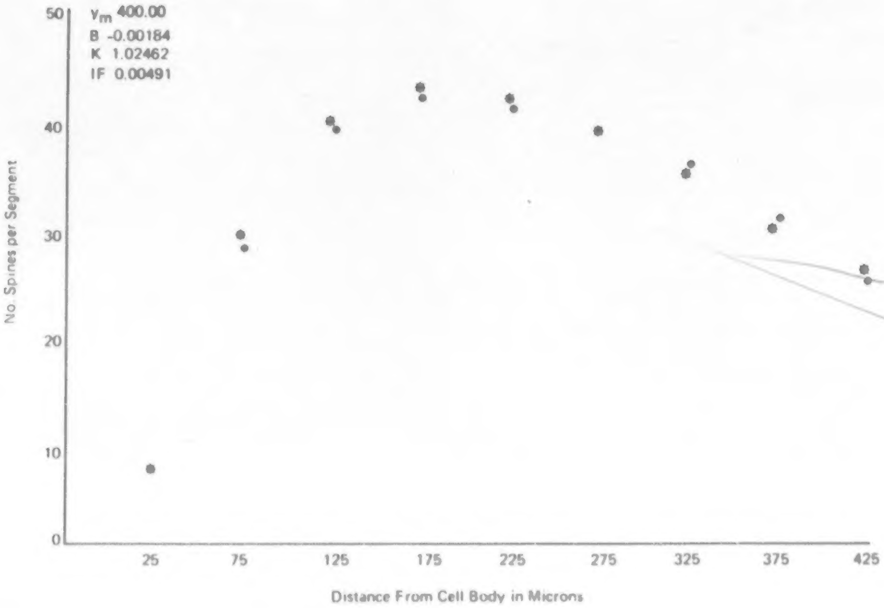

FIGURE 11 Experimental and theoretical distribution of dendritic spines along apical shafts of layer V pyramidal cells of area striata of nine 48-day-old mice raised in darkness. Total dendrites, 81. See caption of Figure 8. Data prepared from IBM 7070 program RV-6803. Chi value 0.152, 8 df, $p < 0.005$.

served in normal mice were significant, except for mice whose eyes were not yet open.

The distribution of dendritic spines in dark-raised mice, then, may be described by Equation 2. Values of IF, B, and K corresponding with the age of the animal can also be described by Equations 3–5, except function $K = f(T)$, which expresses the exponential modulation observed for normal mice and is not presented here. The mathematical model of the distribution of dendritic spines in dark-raised mice appears to be represented by a set of equations similar to Equations 2–5 for normal mice with a series of coefficients homologous to those corresponding to Equations 2–5.[39]

Figure 12 is a three-dimensional reconstruction (by Lison's method[31]) of the distribution of dendritic spines per segment along the apical shafts in relation to age in normal and dark-raised mice from 10 to 180 days old. The reconstruction has been drawn with values of the theoretical distributions given by the computer according to the mathematical model. The distance from the cell body along the apical dendrites is represented by the x-axis; the mean number of spines per 50-μ segment, by the y-axis; and age, by the z-axis. Four sections perpendicular to the z–y plane, corresponding to the distributions at the ages of 10, 19, 48, and 180 days, were used to build these reconstructions.

Comparison of the two reconstructions reveals a great difference in mean numbers of spines per segment that is associated with age. There is a sharp increase in the number of spines throughout the apical dendrites in normal mice between 10 and 19 days, which is related to the opening of the eyes. In dark-raised mice, the numbers of spines, except at 10 days, appear always below the corresponding numbers observed in normal mice. The surface of the reconstruction is rather smooth and does not present a crest at the 19-day level. As in normal mice, there is a continuous decrease in the number of spines on the last dendritic segments from the 19-day level, and the number of spines on segments closer to the cell body increases continuously with age, although not as markedly.

DISCUSSION

Recent observations of Colonnier[8] show that little of the dendritic interspine surface bears presynaptic formations. His results point out that

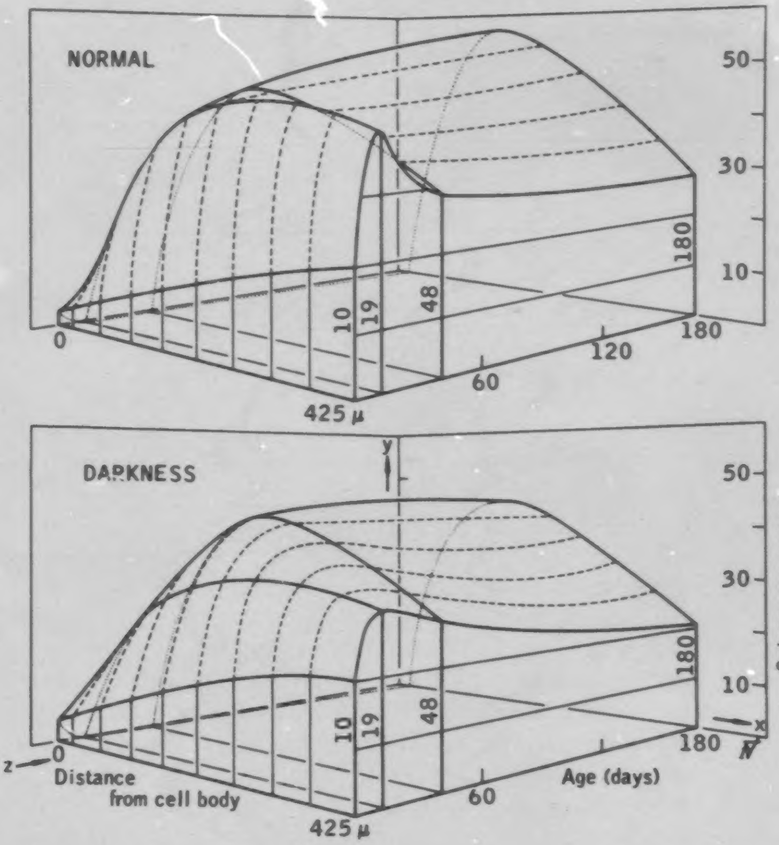

FIGURE 12 Three-dimensional reconstruction of distribution of dendritic spines along apical dendrites and evolution of distribution with age in normal and dark-raised mice. Reconstruction is based on theoretical distributions according to our mathematical model.

the vast majority of synaptic contacts are established on the dendritic spines, and consequently these represent almost the total postsynaptic apparatus of most dendrites in the cerebral cortex. Dendritic spines thus are an exact imprint of the presynaptic dendritic load. Their pattern of distribution, number, and arrangement reflect precisely the distribution, number, and arrangement of presynaptic terminations. This interrelationship has been found particularly advantageous, in that qualitative and quantitative variations from normal spine distribution can specifically indicate the nature and time of visual sensory deprivation, provided that the normal spine frequency and distribution are known. Because the normal spine distribution has been found to be specific for age, quantitative mathematical studies of variations can be used to determine age.

The mathematical analysis of biologic processes is useful insofar as it clarifies the functional relationships between variables. The results may be presented by equations that form a mathematical model. It should be possible to use a model of a biologic process in interpolating and extrapolating to obtain unknown values of variables. This has been one of the purposes of our study of the distribution of dendritic spines and the evolution of the distribution with age in normal and dark-raised mice.

Comparative studies of the mammalian cortex have suggested that the brain is organized at macroscopic and cellular levels according to some invariable laws. These laws may appear extremely complicated, but they might also result in very simple patterns of connectivity that become complex in being repeated again and again. The mathematical model that we have introduced[39,46] demonstrates that the distribution of dendritic spines along the apical shafts of layer V pyramidal cells of the area striata in the mouse follows a mathematical law defined by Equation 2, which is valid for all age groups studied. If we now consider that every dendritic spine supports at least one synaptic connection, it is evident that the patterns of connectivity with respect to apical dendrites might be organized in part according to this law. Furthermore, the functional relationship between the values of the coefficients IF, B, and K and the time, T, defined by Equations 3–5, or their homologues for dark-raised mice, indicates that the evolution of this process follows particular laws represented by these equations.

We have repeatedly found evidence that short axon cells do not connect with apical dendrites at random, but according to the pattern of distribution of the apical dendritic spines.[46] On the one hand, that means that connections may be established not by chance proximity, but according to particular intraneuronal and interneuronal factors that govern cortical organization. On the other hand, in relation to the distribution of spines along the apical dendrites, we have applied program RV-6803 to the data of spine distribution in man obtained by Marin-Padilla[34] and obtained close correlations ($p < 0.005$) with the theoretical distribution formulated by Equation 2 in all his cases. This is an important observation because it constitutes evidence that the distribution of spines along the apical dendrites follows the same laws in man as in the mouse.

Our model has been used to predict the distribution of the dendritic spines corresponding to other age groups not previously studied, as well as to find out the age of an experimental animal from the known spine distribution. This last application might be of considerable practical interest in a wide field of approaches to the study of brain evolution. The distribution of the dendritic spines in the visual cortex and its variations with the age and condition of the animal have been found highly specific, and it is on this specificity that the validity of the application rests. The results were obtained with fairly good approximation to the real age ($p < 0.05$) in almost all our examples. Obviously, exactness would be obtained after addition of coefficient values corresponding to more age groups to provide margins for the natural dispersion inherent in all biologic processes.

In relation to spine loss in enucleated mice, Valverde[44] has stated:

> Minkowski's early observations[36] of transneuronal degeneration in the lateral geniculate nucleus after eye removal, first revealed the existence of a functional dependence of post-synaptically related neuronal structures, from their afferent fibers. These observations, which were later extended through many well-known studies, led to the general acceptance of the notion that, in cases of close or exclusive dependence, the post-synaptic element suffers a process of mild, long lasting and progressive degeneration, which may eventually effect a complete loss of the cell.
>
> An early report by Donaldson[13] on the famous Laura D. Bridgman's case, as well as other reports of cases of human blindness, reviewed by Tsang,[42] alleged the existence of cortical atrophy of the visual area. Many of these studies have been discredited for the lack of adequate controls, but recent experimental observations in Rodents and Lagomorpha[26,28,30,42] proved that removal of the eye has significant effects on the fiber and cell content of the visual area. The evidence so far strongly suggests that transneuronal degeneration is not halted at the first post-synaptic element, but damage in the main afferent supply may set up a progressive involvement of successive neuronal links of the sensory neuronal chain.
>
> Transneuronal degeneration appears, however, difficult to be predicted in terms of severity and time-course. It depends upon at least three main variables: age, species differences, and nuclear formation in question.[35] A quantitative relation exists moreover between the amount of reduction of the afferent supply and the resulting degeneration.[29] In many instances transneuronal effects do not lead to complete degeneration of the post-synaptic element, but only to production of slight structural changes. In this case we should speak of transneuronal *changes* rather than *degeneration* and raise the question, whether or not these changes are similar to structural modifications of neurons resulting from functional demands.

> The diminution of spines observed [in enucleated animals] might represent a transneuronal change of this nature whereby functionless or degenerating specific afferents in layers IV and III would induce the removal of the spines attached normally to these afferents. This assumption is based further on the demonstration that synaptic contacts are not easily broken. Portions of post-synaptic structures remain firmly attached to isolated endings in damaged tissue or in centrifugated preparations.[17,19,20] Colonnier[7,9] showed in the cortex that post-synaptic membranes and spines attached to degenerating terminals are also phagocytosed.

Histologic alterations in the cortical visual centers of mice raised in complete darkness have been studied by Gyllensten and co-workers.[21,22,24] They found decreased mean volume of internuclear material and nuclear size at the age of 1–4 months. The existence of a statistically significant diminution of the mean number of dendritic spines per segment in the apical shafts of layer V pyramidal cells of the visual cortex in dark-raised mice was first reported by Valverde.[43] Changes in the morphology of the dendritic spines of young rabbits subjected to visual deprivation for the first 30 days of life have been described by Globus and Scheibel.[16] Coleman and Riesen[6] showed that stellate cells of layer IV in the visual cortex of cats reared in the dark have smaller dendritic length and fewer dendrites than those of normal animals. All these studies point out, as we have stated,[43] that visual sensory deprivation affects the fine structure of the central nervous system and that some structural changes in nerve cells might occur as the result of experience.

The problem of spine function has been discussed recently by Scheibel and Scheibel.[40] The current point of view is that dendritic spines receive characteristic signal patterns whose spatial and temporal integration might code specifically the function of each cortical neuron. The significance of the diminution of the number of dendritic spines in some experimental situations (e.g., dark-rearing and enucleation) has been discussed elsewhere, with the spines considered as structures capable of reflecting the functional state and the damage of their afferent fibers.[43-45] The distribution of the dendritic spines in dark-raised mice reported here demonstrates that there is a more reduced rate of spine growth after mice open their eyes for all the age groups we have studied. These changes appear to be due entirely to the effects of light deprivation, specifically in the visual cortex; we reported previously[43] that no diminution of spines was detected in other cortical areas when mice were reared in darkness.

There are two important considerations. First, the distribution of spines along the apical dendrites in dark-raised mice shows mean values below normal for every dendritic segment, but the characteristic distribution formulated by our mathematical model is maintained for all age groups. This seems to indicate that the supposed somadendritic factor responsible for the distribution of dendritic spines is not affected by visual deprivation. Second, we have not found evidence of partial normalization of the number of spines in any group of dark-raised mice. Gyllensten *et al.*[24] found slight growth and normalization of the volume of internuclear material and the diameters of cell nuclei in mice after long periods in the dark.

Finally, we cannot yet identify the intrinsic mechanism that produces the diminution of dendritic spines in dark-raised mice. Two explanations are possible: (1) visual deprivation has a transneuronal, metabolic, or other deleterious effect on the spines, so that some of them would be removed; and (2) dendritic spines would not grow normally in the absence of normal visual inputs. Whatever the effect might be, the theoretical interest of our observations is obvious: they may give new clues to the anatomic plasticity of the brain in relation to behavioral and learning phenomena.

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DISCUSSION

DR. SPERLING: Do you think that some spines are present at first and then fall off as the animal gets older? You have a given number of spines at one age and fewer spines in the same position a little later. What happened to them?

DR. VALVERDE: The curves actually crossed the last part of the 180-day-old distribution. We do not know what happened here; it may be that the spines have somehow disappeared.

DR. RIESEN: I might point out that the Scheibels (Exp. Neurology 19:331-345, 1967) have reported shriveled spines, some of which they believe will disappear.

DR. DOTY: What about the basal dendrites?

DR. VALVERDE: We have completed work only on the apical dendrites and are just beginning to work on the basal dendrites, as well as on the dendrites of other neurons. We suspect that our model will fit all spine distribution in most dendrites, but we have to prove that. At present, we have no data on the matter.

DR. BUSER: I have found that the anatomic results would fit some electrophysiologic data of mine.

DR. LINDSLEY: Dr. Valverde, how can you be sure that these fibers are from the lateral geniculate and not from some other source?

DR. VALVERDE: This is, of course, very complex in most mammals. The cortex of the mouse is rather simple. Fibers entering area 17 approach layer IV obliquely. They can usually be followed long enough through the white substance to make sure that they do not come from neighboring association cells. There is another distinction. The fibers coming from the lateral geniculate nucleus are very thick and make complex arborizations, particularly on layers IV and III. Association fibers are much thinner and reach layers II and III primarily.

DR. DOTY: It is well known that the primary afferent fibers coming into the striate in primates can be identified on the basis of their coming in on a slant across the cellular columns, whereas all the intercellular arrangements seem to be directly perpendicular to the surface.

Inasmuch as you are working with the mathematical treatment of your data, you can anticipate and extrapolate them to infinity. What do they predict for a mouse that lives forever? And, a more serious question, in the synapses of, say, the stellate cells on the pyramidal cells, or of the afferent fibers on either stellate or pyramidal cells, do the endings select a particular portion of the dendrite? Are they limited to only one portion, or might an afferent end on both the basal and the apical dendrites?

DR. VALVERDE: Functions $IF(T)$ and $B(T)$, two potential functions relating the coefficients IF and B of the principal equation with the age T, asymptotically approach given values, which we defined as IF_m and B_m, respectively. Consequently, the number of spines tends to stabilize at maturity.

We have been working with a procedure for anterograde degeneration for the tracing of pathways, and have found, as on our Golgi material, that specific afferents arrive at the cortex undivided; as the fibers pass up to layer IV, they make associations with apical and other dendrites. The basal dendrites of pyramidal cells of layer V are below this level, but we believe that the basal dendrites of the layer III pyramidal cells receive terminals from the specific afferents. It is very difficult, however, to stain these specific afferents, and we have to work this on hundreds of sections. We have not yet observed that specific afferents select particular dendrite portions.

DR. DOTY: What about the stellate-cell endings? Do any of them end on both basal and apical dendrites?

DR. VALVERDE: Yes. Although in higher mammals there would be more diverse types of cells, in the mouse we have found at least two definite types. There is a type of stellate cell with axons going up to the superficial layers and synapsing on apicals at the level of layers II and III. The other type makes a very complex axonal arborization, and, in this case, the collaterals of the axon might actually contact both basal and apical dendrites on the same or on other cells.

MERTON C. FLOM

Early Experience in the Development of Visual Coordination

The human visual apparatus is surprisingly well developed at birth, and it proceeds rapidly in its further development. Within hours after birth, the human infant can sustain visual fixation on a stationary target,[14] move his eyes conjugately in the direction of an invisible auditory stimulus,[20] make compensatory eye movements in response to movements of his head,[14] and exhibit nystagmus in response to moving black and white bars of various widths.[3,6,8,15] Within a few weeks, he can pursue a simple moving target with his eyes.[15] And by 7 or 8 weeks, ocular accommodation[9] and convergence[14] can be exhibited. With this early motor development of the eye, there is a coordinate sensory development that involves a remarkable capacity for human perception and resolution.

To what extent does experience affect oculomotor and oculosensory development in the human infant? For this large question we have only small answers. In an albino infant, light is diffusely scattered across the retina, which results in lowered visual acuity. If this continual scattering of light is not remedied at an early age (for example, by means of an artificial pupil), permanent ocular nystagmus develops.[13] Scattering of light and obscuring of the retinal image by congenital cataracts also prevent the normal development of foveal fixation and acuity, even after removal of the cataracts later in life.[19]

The abnormal experience of continually diffuse imagery, as in albinism and congenital cataracts, is blatantly severe and fortunately rare in humans. A less severe and less rare form of abnormal experience is produced by strabismus ("turned eye"). In this condition, the retinal image of the turned eye receives a continuous flow of visual information different from that received by the fixating eye. Visual deprivation may also result from "suppression" of the turned eye's image.

STRABISMUS AND VISUAL ACUITY

Some children with strabismus develop in the turned eye an amblyopia, a reduction in visual acuity that cannot be improved with lenses or attributed to disease. The onset of strabismus normally occurs between birth and 5 or 6 years of age (it rarely develops after these ages), and it is relevant to ask whether the acuity of an amblyopic eye is poorer if the onset of the strabismus is earlier or treatment begins later.

In 1903, Claude Worth[26] reported on 985 children with constant convergent strabismus whom he had examined and followed during the previous 10 years. I have reanalyzed Worth's data and have displayed them in a way suited to the present discussion.

In Figure 1, Worth's data are presented to show how the time of onset of strabismus and the delay in treatment affected the development of acuity. Displayed on the x-axis is the posttreatment visual acuity, ranging from 20/20 to less than 20/200 acuity and including a category for children who lost the ability of steady monocular fixation. Treatment consisted of glasses to neutralize the refractive error, occlusion of the normal eye with a patch or atropine, orthoptic training, and surgery on the extraocular muscles.

Of the children who had onset of strabismus before 1 year of age and were treated immediately, approximately 90% (y-axis) ultimately obtained normal visual acuity of 20/20. If treatment was delayed by as much as 3 months, but no longer, only about 60% or 65% of the children ultimately obtained normal acuity. If treatment was delayed by more than 3 months, none of them obtained normal acuity, few obtained 20/30 or 20/40 acuity, and most had poorer than 20/200 acuity or lost the ability to fixate monocularly with the amblyopic eye.

If the onset of strabismus was between 1 and 3 years of age and treatment was delayed by no more than 3 months, about 90% obtained

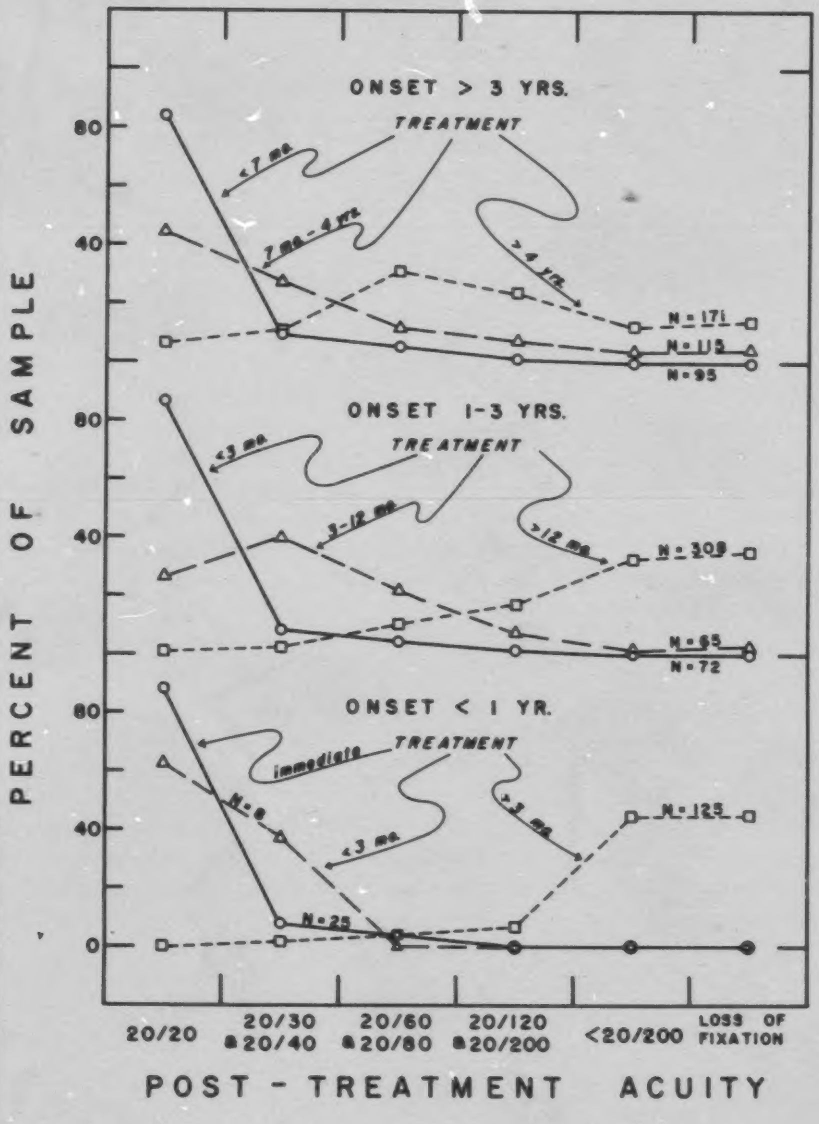

FIGURE 1 Data of Claude Worth[26] on 985 constant convergent squinters displayed to illustrate the influence of the onset of strabismus and delay in treatment on the development of visual acuity.

normal acuity. If treatment was delayed by 3–12 months, a reasonable number of the children obtained normal or nearly normal acuity. If treatment was delayed by more than a year, most of the children did not obtain good visual acuity, and approximately 40% lost the ability to obtain monocular fixation.

If strabismus occurred after 3 years of age and treatment was delayed by as much as 7 months, about 85% of the children obtained normal acuity. Even if treatment was delayed for 4 years and more, many children obtained moderate recovery of acuity, and only a small proportion lost the ability to fixate.

Worth's results show that normal acuity was not obtained in amblyopic eyes if treatment was delayed by more than 3 months after onset at the age of less than 1 year, by more than 12 months after onset between 1 and 3 years of age, or by more than 4 years after onset later than 3 years of age. In short, recovery of acuity in amblyopia was related both to the age at onset of strabismus and to the delay of treatment.

Unfortunately, both Worth and the subsequent editor (Chavasse) of his book[27] made remarks over the years to suggest to others[1,4] a critical age of 6–8 years after which recovery of acuity in amblyopia is supposedly hopeless. The results of Worth's study do not support the idea of a critical age for recovery in amblyopia. Worth's experimental results seem to be forgotten.

Although many doubts have been raised about the validity of Worth's data, for the moment I propose to accept them as valid and to see whether they support the notion of a critical age. Assuming that Worth was able to determine the age of onset precisely and that the time of treatment was relatively independent of the severity of the defect, the data indicate to me no specific age beyond which acuity is irrecoverable. If there is a critical age, it is much earlier than 7 years. Consider Worth's data for the children in whom onset of strabismus occurred before 1 year of age. If treatment was delayed by more than 3 months, practically no children obtained acuity better than 20/200. To emphasize 6–8 years as a critical age is clearly misleading, for Worth's data indicate that treatment begun at 15 months of age is too late for these children.

Since Worth's early study, there have been many reports of acuity improvement in amblyopia when treatment was begun at ages up to 60 years. On the basis of research reported in his textbook and in more than 10 papers on strabismus and amblyopia, Peter[16] concluded

> . . . there is no definite upper age limit in the correction of amblyopia. Until the seventh year, most cases can be restored to normal vision with proper methods; from 7 to 12 the percentage of recoveries is slightly lower, from 12 to 21 years the possibilities are still fair. . . .

In an investigation of 91 amblyopes over 5 years old, Scobee[17] found that 69% obtained 20/40 or better acuity after treatment. Of 34 amblyopes whose average age was 8 years, Dowling[5] reported that treatment led to 76% obtaining 20/40 or better acuity. Kasser and Feldman[12] showed that 31 of 34 adult amblyopes (ages 25–60 years) had improved acuity in the amblyopic eye after treatment.

It is relevant to cite here three representative investigators who have used the recently developed "pleoptics" for treating amblyopia. Bangerter,[2] originator of the term "pleoptics" and director of the world's largest amblyopia treatment center, has reported on some 300 patients as follows (translated from the German):

> In high grade amblyopia . . . the few completely negative cases were limited mainly to children between three and six years of age. . . . The majority of patients who were treated were children between six and twelve years of age; but even among the fifteen and sixteen year olds treatment did not prove hopeless.
>
> The percentage of failures in middle grade amblyopia is small. In cases with low-grade amblyopia the treatment was successful almost without exception. The only fact which deserves to be pointed out is that it was possible to obtain essential improvement in a relatively large number of cases beyond the age of childhood.

Tommila[18] used pleoptics on 137 Finnish amblyopes 6–16 years old and observed that about 60% obtained 20/40 or better acuity and that about one third achieved normal acuity. Girard *et al.*[7] reported that 96% of 71 amblyopes 6–48 years old showed improvement in acuity after pleoptics; 58% achieved a final acuity of 20/30 or better.

The actual success rates in improving acuity in older amblyopes are unimportant here. What is important is the evidence that amblyopia can be improved and even corrected in patients older than 8 years.

NORMAL DEVELOPMENT OF ACUITY

Let us now consider the development of acuity in the normal infant. Many innovative experiments have been done in the last several years to determine (or approximate) the visual acuity of the human infant. In 1963, Weymouth[21] analyzed most of the infant-acuity data reported to that date. He pointed out that the assembled data are too low, and that the median curve (Figure 2) fitted to these data therefore probably underestimates the rapid development of visual acuity in the human infant. The steep initial rise in the median (middle) curve shows that by 1 year of age, the average child has at least 40% of the acuity he will have as an adult, and by 2 years of age, he has at least 80% of the acuity he will have as an adult. Weymouth argued that the 90th-percentile curve (upper) may actually depict the average corrected (with lenses) acuity for children. On this basis, one could expect the average child to have developed about 73% of his adult acuity by 1 year of age, and about 92% of his adult acuity by 2 years of age. In any case, it is clear that a child's visual acuity develops remarkably rapidly and reaches nearly its maximum by about 2 years of age.

Somewhat related to our discussion on the development of acuity and amblyopia in human infants are the experimental results of Hubel and Wiesel.[10] They cut loose the right medial rectus muscle of four

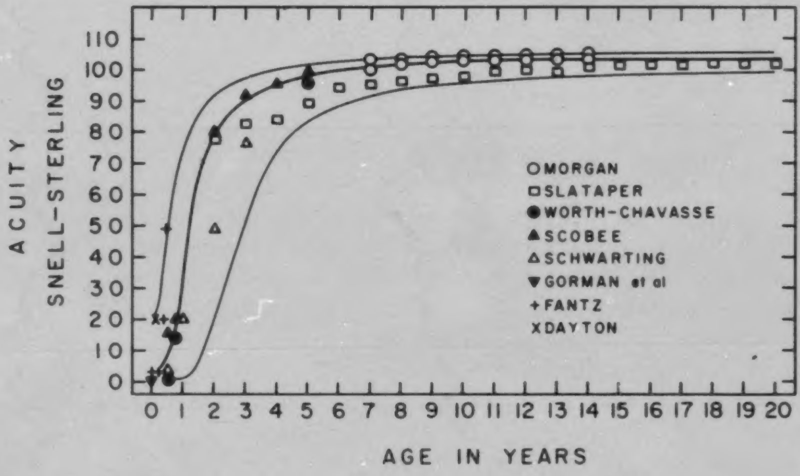

FIGURE 2 Composite data on visual acuity in children. The central heavy line represents the median acuity; the upper and lower lighter lines represent the 90th and 10th percentiles, respectively. (Reprinted with permission from Weymouth.[21])

kittens between 8 and 10 days after birth, thus creating a right divergent strabismus. After 3 months to a year, these animals exhibited no behavioral visual defects to suggest an amblyopia in the "turned" eye. This failure to develop an amblyopia in the deviated eye may have resulted from the cats' frequently observed fixation with this eye. In any event, the only structural change obtained was a substantial decrease (by one fourth) in the number of striate cortex cells that could be driven from input to both eyes. They concluded that the lack of synergy in the input from the two eyes—as created by strabismus—caused a disruption in the neural connections that subserve binocular interaction. These results bear more on the problem of abnormal binocular directionalization (e.g., anomalous retinal correspondence) than on the problem of amblyopia. Other experiments by Hubel and Wiesel,[11, 22-25] in which one eye of young kittens was sutured shut or was covered with a translucent occluder, produced marked histologic changes in the lateral geniculate nucleus, physiologic changes in the striate cortex, and some perceptual impairment. These latter experiments with form deprivation in cats speak more relevantly to the problem of the development of acuity in human infants, but care must be taken in extrapolating these results from cat to man.

Several points emerge from the preceding discussion. First, the oculomotor and visuosensory (visual acuity) systems are nearly fully developed in the human infant by about 6 and 24 months, respectively.

Second, embarrassment of information to one eye during childhood can lead to amblyopia. If the embarrassment occurs during the developmental period of visual acuity (birth to 2 or 3 years), then the ensuing amblyopia may include some portion due to impairment of normal development. Embarrassment after this period probably results in a genuine loss of function that was previously present. There is no evidence that 6–8 years is a critical age beyond which recovery of acuity in an amblyopic eye is impossible. On the contrary, there is considerable evidence that many amblyopias can be improved or corrected in late childhood and even in adulthood. If there is a critical age for the treatment of amblyopia, it is more likely to be 1–2 years for children who have experienced visuosensory embarrassment in infancy.

I think that we can conclude from the observations discussed that, if it is considered advisable to provide suitable visual experience to assist the development of the visual system, this experience should occur quite early in a child's life—probably before 1 or 2 years of age.

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DISCUSSION

DR. KAGAN: In Worth's data, do we have a problem of whether the doctor may have decided to treat only easy cases early? Is it possible that the start of treatment depended on the severity of the strabismus? If he said, "If it is a hard case I won't treat it," then your conclusion would not be possible.

DR. FLOM: I think Worth had a predisposition for early treatment and tried, wherever possible, to give treatment early. Those instances where treatment was not instituted early probably resulted from parental noncooperation—that is, they did not bring the child back for treatment or moved away and came back several years later. I think that is usually the reason for delayed treatment, but there may be some confounding of this, as you indicated.

DR. KAGAN: In either case, the samples of children are not evenly distributed.

DR. FLOM: To justify the conclusion that emerges from Worth's results, one must presume that the time of instituting treatment was relatively independent of the severity of the amblyopia.

DR. LUDLAM: I think there may be an even greater confounding factor: the identification of the age of onset. It was said back in 1896 that one could believe almost anything except the age of onset, because mothers traditionally think that children got this on their own, and not in association with the mothers. There are all kinds of emotional situations involved, so that parents traditionally state the age of onset of amblyopia or strabismus as much later than it really is.

DR. ALPERN: What is the evidence that albinos fitted with artificial pupils at an early age develop normal acuity?

DR. FLOM: I did not mean to suggest that *normal* acuity will necessarily develop in an albino if he is fitted with an artificial pupil at an early age. I was referring to the work of Kestenbaum, which I cited, wherein the use of pinhole spectacles in an albinotic infant before 4 months of age (the age at which development of the macular area is normally completed) led to reduced nystagmus and *improved* acuity. Meloan has reported similar results (Amer. J. Optom. 28:435–437, 1951). Dr. Monroe Hirsch (Berkeley) and Dr. Leonard Apt (UCLA) have recently collaborated in providing pinhole spectacles for an albino infant, but their results have not been published. Artificial-pupil contact lenses (with opaque iris and scleral sections) have been used by Edmunds (Arch. Ophthal. 42:755–767, 1949) to increase acuity of older children and adults with albinism.

DR. ALPERN: The histologic evidence of albinism is absence of a foveal pit, and it is surprising to me that merely fitting with an artificial pupil will allow normal vision to develop.

DR. FLOM: It is true that cases have been reported in which the fovea was ill-formed or lacking, but the absence of a well-formed fovea is not invariable (S. Duke-Elder, System of Ophthalmology. Vol. III, Normal and Abnormal Development. pp. 314–1190, Part II, Congenital Deformities. St. Louis: C. V. Mosby Co., 1964). Kestenbaum's idea is that the use of an artificial pupil in infancy aids the development of the fovea in albinos (*Clinical Methods of Neuro-ophthalmologic Examination*, New York, Grune and Stratton, 1961). Edmunds, however, has proposed that overstimulation of the fovea in albinos leads to atrophy of an otherwise normal fovea.

DR. DOTY: I have the impression that Hubel and Wiesel did not get an irrecoverable amblyopia except when they used the plastic occluder on one eye. If that is the case, then there is some very important information here of relevance to the human situation. If you produce strabismus in a cat by cutting the muscle, then you do not get amblyopia. But you do get amblyopia in the same species by playing with the process as it affects the central nervous system. This would suggest that the amblyopia does not arise simply from noncongruence, but can also arise from some problem in the lack of visual acuity itself. Are there any human cases that are comparable to the Hubel-Wiesel situation, in which the amblyopia arises from a purely physical difficulty with the peripheral muscles, rather than a difficulty in the central control mechanisms?

DR. FLOM: I am not sure that the loss of vision that occurs in kittens as a result of light deprivation is amblyopia—it is a loss of vision. In a population of amblyopic persons, about one third will have strabismus alone, one third a substantial difference in refractive error between the two eyes (anisometropia), and one third both (data to be published). That covers approximately 99% of the total sample of amblyopes.

It appears that the two associated conditions in amblyopia are a deviant eye, which produces a lack of synergy in the two visual inputs, and one defocused retinal image. Each of these conditions has, of course, its counterpart in the Hubel and Wiesel studies. Defocusing of the retinal image in one eye is analogous to the experimental situation of placing half a Ping-Pong ball over the eye, so that diffuse light but no image falls on the retina. Their experiment[10] in which they operated on the medial rectus muscle is related to strabismus.

DR. DOTY: Not necessarily, because the origin of that strabismus may be a central defect or a problem in neuromuscular control.

DR. FLOM: There are types of strabismus that are purely peripheral in origin—that is, they are due to abnormalities of the muscles or ligaments.

DR. DOTY: What happens in those cases? Are they equally amblyopic?

DR. FLOM: I cannot say. I do not think anyone has looked at the distribution of amblyopia in two different classes of strabismus. That would be worth looking at.

WILLIAM A. MASON

Information Processing and Experiential Deprivation: A Biologic Perspective

Man's capability in the living world as a seeker and user of information is undeniably unique. Of all his attributes and skills, in fact, most people are likely to agree that man's ability to process information most clearly sets him apart from the rest of the animal kingdom, giving particular weight to language and other cognitive skills that no other creature can equal. It is a common belief that the abilities to speak, to read, to plan, and to cipher are distinctively human achievements.

However, even if we grant that man has exceptional talents as a seeker and user of information, we must still concede that he is scarcely more than an impressive newcomer on the information-processing scene. The need to know about the world and to act on information received are far older than man–older, indeed, than behavior itself, since behavior has emerged in response to these demands. It seems most likely that man's achievements in dealing with information are built on ancient foundations that he shares with many humbler types. Accordingly, our appreciation of the special skills of the human organism as an information-processing system and our understanding of the disabilities to which this system is liable will be improved if they are considered within a broad biologic perspective. Such, at least, is the viewpoint of this presentation.

Other portions of this volume deal with the effects of experience on particular organs and processes in animal subjects. In contrast, my interest here is directed toward a broader set of questions. The primary concern will be with the organism as a whole, viewed as an information-processing system. The thesis to be developed can be summarized briefly: All organisms have evolved within an environment that is stable when viewed broadly and yet on closer examination is seen to be subject to continuous change. Behavior has emerged as an important means of detecting and adjusting to change. To the extent that all behavior is organized with respect to environmental regularities, contingencies, and constraints, the structure of behavior is necessarily isomorphic with the structure of the environment. In this sense, all behavior can be considered as a manifestation of a representational process (schema). "Instincts," viewed as more or less fixed sequences of action in response to particular stimuli (releasers), can be considered to constitute a special case of such representational processes or schemata. Schemata are inherent, species-characteristic modes of organizing experience, but there has been a general trend in mammalian evolution for schemata to become more open, more responsive to variations in environmental input. As would be expected, openness is greatest in species that are the most accomplished and versatile users of information, and it is those species whose behavior most clearly reflects variations in individual experience. In the more advanced species, early environmental restriction will affect schemata in two ways: It will impede their complete development and elaboration by withholding "essential" classes of information to which the individual (as a representative of a particular species) is selectively "tuned," and it will lead to the development of seemingly aberrant or distorted schemata that are actually reflections of (or isomorphic with) the peculiar properties of the rearing environment.

ORGANISM AND ENVIRONMENT

The basic relationship we will be concerned with, of course, is the relationship between the organism and its environment, and it will be helpful to deal with this question first in rather general terms. This relationship is obviously fundamental—not only in behavioral research, but in the whole of biology—but does not imply that the more complex behavioral attributes of man can be reduced to terms appropriate to a rat or a frog. It does imply, however, that even the most complex behavioral attributes have an evolutionary history and serve a biologic purpose.

Man has to cope with the same inexorable demands as the monkey or the mouse—maintaining proper body temperature and oxygen tension, finding enough to eat, procreating, providing for the young. These are as essential to our own survival as a species as they are to the survival of any other biologic group. What is important is the obvious fact that all of us, man and animal alike, are the products of our environments, and we bear the stamp of our origins on every aspect of our structure and function. The environment implies the kind of organism that it can sustain, and the organism implies its appropriate environment.[8] The information that an organism can register and how it gets this information and uses it are determined by the exigencies and constraints of the environment in which it has evolved. One can look upon the organism as a biologic system that exists for the "purpose" of achieving a workable fit between its vital functions and its environment and that continues to exist because it fulfills this purpose with some degree of success. That is the essential idea behind Darwinian fitness.

Clearly, however, to speak of the environment in this way—as though it were a static entity—is merely a convenient fiction. As we all know, the environment is not a "thing," but a process, and change is the rule. No environment remains the same for long, and no two environments are ever precisely alike. There are regularities, to be sure; without a large measure of stability, life would be impossible. But stability is only a matter of degree, and even in the most nearly constant of environments, some element of caprice is always potentially present. All successful organisms possess some ability to cope with the reality of change, some way of altering their functions in accordance with changing circumstances.

The necessity of coping with change has given rise to countless evolutionary innovations, and among these, behavior must certainly be included as one of the most significant. Its peculiar virtue is flexibility. "Behavior" implies the capacity to select from the total array of environmental energies particular "relevant" segments (for example, from the electromagnetic spectrum), to encode this energy, and to use the encoded information as the basis for selecting some response. The "apparatus" of behavior thus has as its main business the evaluation of environmental contingencies and the selection and elaboration of appropriate courses of action. An amoeba's movement toward a weak stimulus is a reaction to the "probability" that a suitable food object will be encountered.[26] Likewise, a monkey's leap from one tree to another implies a statistical "faith" in the weight-bearing potential of tree limbs, the reliability of distance cues, and so on. From this point of view, all behavior points forward; it has a predictive or probabilistic component.[3,4] Even the simplest-behaving organism is always of necessity playing the odds—using information received to initiate some program of action as the most likely to result in success.

REPRESENTATIONAL PROCESSES

How information is received and organized, the form in which it is stored, and how it comes to be tied in with specific acts are subject to debate. Until recently, the most heated arguments revolved around stimulus-response (S-R) versus cognitive views of learning. Stimulus-response theories grew out of reflex-arc conceptions of learning, especially the work of Pavlov, whereas cognitive theories are most heavily indebted to the organismic views of Gestalt psychology and of Piaget. It is becoming clearer every day, however, that the issues that separate those positions are more often semantic and methodologic than substantive. The main virtues of the S-R formula are that it is simple, explicit, and easily translated into operational terms. It is the working language of the scientist at his bench. But no one could claim that the S-R formula in its primitive, unadorned form can handle the complexities of intellectual life in the more advanced species. And with every concession to that fact—concessions expressed by postulating hypothetical processes intervening between stimulus and response—the S-R position and the cognitive positions move closer together.

The shape of the final rapprochement, however, is not our concern. What we are seeking is a way of characterizing the information-processing activities of nonhuman forms that seems to fit the facts. And I believe that we are constrained by the evidence now available to recognize that all information-processing activities occur within the framework of existing systems. The sequence consisting of the selection of stimuli, the encoding and storage of information, and the elaboration of specific actions is from beginning to end a highly organized process.

How we refer to this process—as instinct, habit, set, expectancy, engram, cognitive map, plan, program, or schema—is determined largely by the complexity of the phenomenon being considered, by our knowledge of its history, and by our preference for "analytic" as opposed to "synthetic" terms. It boils down to a matter of scope and personal taste. The term that will be used here is "schema."

Schemata are by no means the exclusive property of more advanced animals. On the contrary, they are very widely distributed within the animal kingdom. They play a major role in the performance of biologically essential tasks, such as mating, care of the young, filial responses, reactions to predators, and pursuit of prey; and they provide the basic groundplan according to which behavioral development must proceed. They are the major vehicles for maintaining an acceptable fit between behavior and ecology. The evolution of schemata has occurred within a particular array of environmental energies. They have been subject to the same kind of selection pressures as any other organismic functions; therefore, they necessarily reflect the regularities, contingencies, and constraints that the environment imposes. In this sense, they can be regarded as functional representations of the environment.

The frog waiting patiently for his supper affords one example of such an organized representational process in one of the humbler vertebrate forms. The frog is equipped with his schematic fly. To be sure, the schema seems primitive by our standards, because it is such a poor representation of flies as we know them. For the frog, a fly is any small object that moves into its visual field, no matter whether it is edible, and the fly-catching program is activated by that movement. Under most circumstances, such a program accomplishes its purpose effectively—flying pebbles, after all, are something of an anomaly in the frog's natural environment. But one of the limitations of the program is that it is unable to recognize flies if they are not moving. This creates a problem for frog fanciers, in that the frog can starve to death surrounded by immobile but perfectly nutritious flies, and steps must be taken to animate its diet. The frog is not entirely without resources for modifying its prey-catching schema in the face of new information, however; if a hairy caterpillar is substituted for the fly, the snapping response is inhibited after a few feeding attempts, at least temporarily.[21,26,37]

The situation does not appear to be vastly different at the outset for animals much closer to man. The newborn monkey begins life with a schematic mother, which is a far cry from monkey mothers as we know them. In the beginning, the defining properties of "mother" are almost entirely tactile, and virtually any claspable object can serve as a mother substitute.[18] In contrast with the frog's schematic fly, however, the monkey's primitive schema undergoes a rapid and progressive enrichment as development proceeds. As physical attributes and their arrangement in time and space become associated with a single source, "mother" emerges as an entity; she acquires object status. Eventually, she is recognized at a distance and from various angles of regard. Her distinctive physical attributes combine with a great number of more dynamic functions that help to define the relationship between mother and child. The growing monkey ultimately arrives at a conception of "mother" that in subtlety and complexity must come close to our own.

PHYLOGENETIC CONTRASTS AND TRENDS

It is clear that, as species become more "intelligent," they also become less "instinctive." The contrast between the schemata of the frog and the infant monkey suggests how this relationship may have come about. Presumably, all schemata are innate to the extent that they exist in every normal individual as inherent species-typical dispositions toward organizing experience in particular ways. At the same time, however, all schemata are probably "open" in some degree. Those which are less open are susceptible to little more than minor "tuning" in response to altered environmental input, whereas the more open schemata are subject to extensive modification and may even remain functionally incomplete in the absence of appropriate input. And, of course, it is precisely the species that we consider most "intelligent" that display the greatest degree of openness.

In other words, in the more advanced biologic forms, limited but highly stable systems for finding, encoding, and acting on information have been exchanged for increased behavioral plasticity. Relatively closed and invariant schemata are highly adaptive in a stable environment, and this advantage has been sacrificed to achieve greater flexibility in adjusting to change. "Instincts" (i.e., primordial schemata) have not become lost in evolution, but they have become progressively more open, and the result of this transformation has given rise to what we refer to as "intelligence."[22,23,25,41] The advantages of this development are obvious, and so are some of the risks. Increased openness of schemata amounts to a degeneration or dissolution of the organization of instinctive patterns, and this change will be adaptive only if some provision is made for creating behavioral stability by other means. And it is at this point, of course, that an improved capacity to deal with information becomes critical.

The functional changes that accompany this improvement are fairly well agreed on, even if the underlying mechanisms are obscure, and I will describe them briefly here (for documentation, see, e.g., Harlow,[15] Lashley,[23] Maier and Schneirla,[26] Nissen,[35,36] and Warren[43]):

Increased sensory differentiation. Within any given situation, the range of effective stimuli increases, as does the ability to respond selectively to different patterns or configurations of stimuli. There is increased effectiveness of complex perceptual relations (such as figure–ground, salience, and congruity) and of the various perceptual illusions. Presumably, the same process is also involved in some types of insightful problem-solving and detour behavior.

Increased responsiveness to stimulus change. This is expressed in the elaboration of attentional mechanisms (orienting reflexes, scanning, observing responses, visual curiosity) and in the tendency to respond to novel stimuli with a wide range of spatial adjustments resulting in approach (exploration, manipulation, play, aggression) or withdrawal (timidity, fear of novelty, flight).

Increased ability for sensory integration. This is expressed in improved capabilities for linking together information from different modalities, or from the same modality at different times, for abstracting, generalizing, and elaborating rules and strategies on the basis of experience.

Increased differentiation of motor acts. Motor patterns become more highly differentiated and particulate (e.g., ability to move each digit independently versus opening and closing hand as a whole) and motor sequences become "looser" (e.g., ability to locomote using a variety of postures and gaits).

The organism that emerges from such evolutionary developments embodies a superior information-processing system. It is better equipped to select and analyze current information from the environment, to integrate it with previous information, and to elaborate a plan of action. And it has the motor refinements to translate this plan into concrete achievements. Such an organism is opportunistic and stimulus-seeking. Its behavior has become something more than a mechanism for dealing with imposed changes in environmental input. Behavior now has the additional role of creating variable input, which is ordered and organized as it comes in. The cumulative effect of the information it receives is the development, enrichment, and elaboration of schemata. The essential framework for this development is present from the outset.

If this view is correct–if it is true that primitive schemata provide the basic framework within which and on which experience operates–then the deprivation experiment can be viewed as one method for investigating this framework. Withholding information early in life does not eliminate the framework or necessarily prevent its subsequent elaboration. Schemata may continue to develop under conditions in which the impetus and constraints that influence normal development are changed radically. The result is not behavioral disorganization, but a form of organization that reflects the particular circumstances of the rearing environment.

EFFECTS OF DEPRIVATION

The implications of this view will become clearer when we consider the effects of deprivation on behavioral development. Particular attention will be given to research in which animal subjects, chiefly monkeys and apes, are raised under conditions of general environmental restriction. It is important to recognize that we will be dealing with a form of deprivation that is not limited to a particular sensory modality, but with a radical distortion in normal ecology. The animal is removed from its mother at birth and raised alone in a cage that permits it to see the nursery environment and other individuals or in an enclosed cage that curtails even that kind of stimulation. If we compare the rearing environment of such an animal with that of an individual growing up in the wild, the differences are enormous. Obviously, such deprivation cuts across the entire spectrum of normal environmental input.

As expected, the behavioral consequences of such a distortion are massive and widespread. Indeed, a primary purpose of much of the work that has been done thus far was to sort out the damage. The aim has been to provide a general overview of the range of behaviors that are affected and to map out some of the more obvious relationships between rearing conditions and development. The emphasis has been problem-oriented and descriptive, rather than process-oriented and analytic. Because of this emphasis, we have not gone very far toward a precise understanding of the sources and nature of specific psychologic disabilities. But we are gaining a clearer view of nonhuman organisms as information-processing systems and of the effects of environmental conditions on the development and functioning of these systems.

It has become clear, for example, that early restrictions on experience produce a predictable syndrome of effects whose major elements have been described for rats, guinea pigs, cats, dogs, monkeys—all mammals, in fact, that have been examined. The deprivation syndrome includes not only deficiencies (functional "gaps" in the behavioral repertoire of deprived animals), but aberrations, behaviors that are not seen in nondeprived animals. There is thus the suggestion of a common mammalian response to restricted environmental input that transcends differences among species and minor variations in rearing conditions. Moreover, there is the implication that developing mammals have similar requirements for information and similar propensities for processing information; animals display not only similar deficiencies, but also similar aberrations, when information is withheld.

It has also become clear as the result of recent research that variations within the broad framework of the deprivation syndrome tend to follow phyletic lines. An animal does not abandon its species-typical modes of processing information, even under extreme conditions. In general, one can say that the closer the phyletic affinity between two individuals, the greater the likelihood that they will resemble each other in their specific behavioral reactions to deprivation, even when the reactions are apparently maladaptive and are qualitatively distinct from behaviors shown by nondeprived members of the species. Such species-typical variations within the deprivation syndrome provide strong support for the view that the selection and processing of input occurs within the framework of existing semiautonomous systems.

Stereotyped Motor Acts

The interplay between environmental input and intrinsic schemata can be illustrated by the stereotyped motor acts that develop in nonhuman primates raised apart from their mothers. The most prominent of these acts are nonnutritive sucking, self-clasping, and body rocking (for review see Berkson[1]). An infant monkey or ape raised alone in a bare cage fails to encounter normal outlets for clinging and sucking and hence comes to direct these responses toward itself. In a limited sense, it develops its maternal schema out of the most accessible and appropriate material that is available, which happens to be its own body. This interpretation is plausible for thumb-sucking and self-clasping, both of which have normal counterparts in patterns that are directed toward the natural mother. The third pattern, stereotyped rocking, has no obvious parallel in the normal mother–infant relationship, but we have recently obtained presumptive evidence that it, too, can be considered a "substitute" response to the deprivation of stimuli ordinarily provided by the mother. If infant rhesus monkeys are raised with stationary surrogates of the sort used by Harlow, most will develop stereotyped rocking as a habitual pattern. If they are raised with the same device, except that it is motorized to move up, down, and around within the cage, stereotyped rocking does not develop, presumably because the robot surrogate simulates the kind of input that the natural mother provides as she carries the infant about. In other words, I am suggesting that stereotyped rocking of the rhesus monkey is a manifestation of a primitive schema working itself out on the basis of reduced environmental input.

The organizing effects of intrinsic schemata on motor patterns are evident not only in the early stages of postnatal life, but throughout development. Isolation-reared monkeys display self-biting and self-stimulation of the genitals, and these self-directed behaviors become progressively more frequent as the animals enter adolescence, the period in which social aggression and sexual interaction assume prominence in nondeprived monkeys.[5] The most compelling evidence of the guiding role of schemata in later development, however, is provided not by experientially deprived animals, but by a mother-reared monkey that was deprived of normal effector mechanisms as the result of a congenital malformation. An infant rhesus monkey with a thalidomide-induced deformity of the forelimbs not only walked bipedally, a behavior that is infrequent in normal animals but can be seen on occasion, but used its feet to groom its mother–a pattern that to my knowledge has never been observed in an intact monkey, an "invention" in response to very special circumstances.[24]

Arousal Effects

Another reliable consequence of early environmental restriction is a tendency toward fearfulness or heightened emotionality. The behavioral expressions of this effect are varied and depend on the particular species and the situation in which the animal is observed. Motor discharge may take an exaggerated form, as in seizures, tics, whirling fits, or frantic running; or it may be minimal, as in crouching, freezing, or falling into syncope or sleep. The general picture is one of behavioral disorganization or the activation of organized patterns that are inappropriate to the situation.

What is the source of these extreme emotional behaviors, and what implications do they suggest for the information-processing activities of deprived animals? The immediate source, it would seem, is any large increase in stimulation over that to which the animal is accustomed, and the range of stimulation to which the deprived animal is accustomed is, by definition, narrow. Seizures, tics, and freezing are most likely to occur in response to novelty or change, presumably because an isolation-reared animal is overwhelmed by the sudden increase in stimulation and has no other means of coping with it.

Plausible as this explanation may seem, however, it does not go far enough. In many experiments, the test situation is also new to the controls, and initially they do indeed show some signs of fear. But they are not overwhelmed; their reaction is less intense than that of isolation-reared animals, and it diminishes more rapidly. A deprived animal's response is often entirely out of line with the degree of novelty or change. For example, a chimpanzee raised in an enclosed cubicle may wait many days before it makes its first hesitant contact with a small and entirely innocuous object, such as a matchbox or a block of wood. If one were to take every precaution to ensure that "novelty" were equivalent for wild-born and restricted animals, as by constructing an object from exotic materials, there is no reason to doubt that the wild-born animals would still contact it sooner than the restricted chimpanzees.[33] The point is that we need know only that one animal was raised in isolation and another was not to predict how their reactions will differ toward any small inanimate object. A detailed knowledge of their individual histories is not necessary and in fact would probably add very little to the accuracy of prediction. Knowing whether or not a specific object has been encountered previously is less important than knowing that there have been many such encounters in the past. It is a question not of whether an object is more novel to one animal than to the other, but of the kinds of reactions that novelty produces.

A deprived organism is ill-equipped to deal with unfamiliar input, with complexity, and with change. It is deficient in ability to assimilate new information, an ability that a normal animal has been exercising almost continuously from early infancy. Excessive arousal in response to novelty or change can thus be considered the result of a breakdown in the processing of information. It is caused by an overload on a system whose development has been curtailed by restrictions on early experience.

A similar view has been advanced by Melzack and co-workers.[30-32] They propose a neurologic model in which sensory input is conceived as a two-part process. Melzack believes that neurophysiologic data point to two classes of afferent fibers that can be distinguished on the basis of conduction rates. The fast afferent fibers are believed to activate central nervous system processes (phase sequences) subserving memory, attention, and similar functions. When activated, these processes cause a feedback to lower synaptic levels, where they can inhibit, facilitate or otherwise modify the input patterns of the more slowly conducting afferent fibers; in this fashion, they exert active control over the selection of information. As the result of sensory restriction, the development of these central nervous system processes is impaired. The selection of relevant information and the filtering out of irrelevant information are faulty, and the loss of filtering leads to diffuse bombardment of the central nervous system, one consequence of which is excessive arousal. This state of affairs can trap a deprived animal in a vicious circle. Lack of experience engenders excessive arousal, and excessive arousal disrupts the activities of the very mechanisms in the central nervous system whose further development is required for orderly and efficient processing of information. To return to the terminology of earlier sections, an advanced organism that has been able to develop only a limited number and variety of environmental schemata because of restrictions on early experience may be hindered by excessive arousal from overcoming this deficiency in later life, even when adequate environmental opportunities are provided.

Recent experience leaves no serious doubt that this is a real possibility. The heightened emotional responsiveness of the experientially deprived animal is, for all practical purposes, a persistent disability. It can be ameliorated by subsequent treatment, to be sure,[11] but it can probably never be entirely overcome. The electroencephalograms of isolation-reared dogs show evidence of high arousal for at least 6 months after they are released from restriction, and behavioral indications of increased arousal persist in monkeys and apes throughout years of exposure to the normal laboratory environment.[5,6,29,32,39]

Basic Cognitive Skills

Certainly the strongest support for the thesis that high arousal exerts a disruptive effect on information processing is the poor performance of restricted animals on learning tasks. Isolation-reared dogs are inferior to controls on spatial-orientation tests, on delayed-response problems (in which an animal is shown food and required to locate it after an imposed delay), and on the reversal of a previously acquired discrimination response (in which an animal must select a previously unrewarded cue).[11,31,42] They are also slower to inhibit a response that no longer leads to reward.[31]

Similar data have been reported for chimpanzees. Davenport and Rogers[6] investigated delayed-response performance in animals raised in isolation for the first 2 years of life. The chimpanzees were 7–9 years old at the time of testing and thus had exposure of at least 5 years to the normal laboratory environment, including experience in group living. Although one might assume that this intervening experience would be sufficient to overcome any early cognitive deficits, the results quite clearly show otherwise. At all levels of delay, the performance of the restricted chimpanzees was markedly inferior to that of wild-born animals, and the differences on the longer delays persisted throughout testing. Data on response latencies strongly suggest that attentional factors were important. At the longer intervals especially, the restricted animals were slow to respond at the end of the delay period, suggesting that they had difficulty maintaining a consistent orientation toward the relevant aspects of the experimental task.

The same subjects were also compared on a discrimination task.[7] The procedure followed the learning-set paradigm developed by Harlow,[16] in which a series of two-object discrimination problems is presented, each problem for a fixed number of trials. As Harlow showed, the facility with which such problems are solved improves with experience, until eventually the more proficient animals are capable of near-perfect performance on the basis of a single information trial. This ability is attributed to the formation of a "discrimination learning-set." The chimpanzees were trained on a total of 438 four-trial object-discrimination problems. Although both groups showed unequivocal evidence of developing learning-sets, the restricted group was inferior to the wild-born group. A significant factor in this inferiority was the tendency to persevere in the selection of a previously unrewarded object.

Contrasting results have been obtained from similar tests performed with rhesus monkeys. Harlow and associates report no differences in delayed-response or learning-set performance between monkeys raised from birth in open-wire cages and monkeys raised as total isolates for the first 6 or 12 months of life.[17, 38] It should be emphasized, however, that all the monkey groups were born and bred in laboratory cages (in contrast with the chimpanzee study, which included a wild-born control group), and it may be supposed that none of the rearing conditions was sufficiently "enriched" to provide for full intellectual growth.

This possibility seems most likely in the light of recent findings obtained at the Delta Center by Mr. Paul Anastasiou. Two laboratory-reared groups of adolescent rhesus monkeys (raised with moving and stationary surrogates), an adolescent jungle-born group, and an adult jungle-born group were tested on 40 different tasks. The problems were designed to assess a variety of cognitive skills, ranging from relatively simple and straightforward visual search to solution of complex puzzle devices. The results demonstrate that both wild-born groups were unequivocally superior to either laboratory group; indeed, the best of the 14 laboratory-reared subjects was essentially no better than the worst of 13 wild-born monkeys.

Fuller, on the basis of his studies of dogs, considers that "behavioral deficits following isolation result more from competing emotional responses than from failure of behavioral organization during isolation or from loss of established patterns."[11] Although heightened arousal probably played a significant part in the performance deficits that have been considered here, it is well to keep in mind that its precise role remains to be established. Furthermore, whatever its role, it is likely that arousal level is only one of several factors that affect the performance of isolation-reared animals. For example, there is convincing evidence that some deficiencies are the result of withholding specific learning opportunities or classes of information.

Specific Functional Relations

It is by now firmly established that some level of stimulation is required for the full structural and functional development of the peripheral receptors and their associated neural systems. This question is fully reviewed elsewhere in this volume and will not be pursued here. The present concern is with an organism that may be assumed to have had a sufficient variety and amount of visual and auditory stimulation to preclude gross neuropathology and to have had the opportunity to form basic sensorimotor coordinations of the sort investigated by Held and co-workers.[19,20] Given such an organism, we are concerned with the extent to which its ability to process information is influenced by specific input features of the rearing environment. How clearly is information received (or withheld) early in life reflected in later behavioral capabilities?

Some of the first evidence suggesting a rather close correspondence between early information input and later performance was obtained with rats. Animals raised in cages with triangles and circles on the walls were shown to be superior to controls in learning to discriminate between these forms as adults.[13] This superiority was maintained (and in some experiments even enhanced) if the test stimuli departed slightly from those to which the animal was exposed in its living cage. Transfer effects were no longer obtained, however, when the differences between the familiar stimuli and the test patterns were extreme.[9,10,14] It thus seems clear that "casual" exposure to specific shapes during the rearing period will not only facilitate recognition of these patterns when they are later encountered in a discrimination-learning test, but will also lead to the establishment of some form of generalization "gradient." It becomes easier to recognize new stimuli that merely resemble the familiar pattern.

The range of stimuli that are responded to as equivalent might be expected to depend on early experience. This question has recently been reviewed by Ganz.[12] Ganz distinguishes between the neural organization of information and the control that information exerts over behavior. On the basis of his own careful studies of hue generalization in light-deprived monkeys and the work of other investigators, he concludes that some organization is innately imposed on stimulus input. The scaling of sensory dimensions occurs before any stimulus experience. This is inferred from the finding that an animal that has been exposed to only one hue value will respond to it on stimulus-generalization tests more than to other hue values. But the generalization gradient is "flat"; the responses to the familiar hue and to the test stimuli are similar. This is viewed as evidence that the dimension as a whole has not developed effective control over behavior.

How is such control acquired? Ganz argues that control is achieved through a history of differential reinforcement within the appropriate stimulus dimension. Thus, an animal must experience reward in the presence of one value of the hue dimension and the absence of reward in the presence of a different value of the same dimension. As the result of such experience, the generalization gradient becomes steeper; the animal differentiates more sharply between the familiar stimulus and the test stimuli. Differential reinforcement does not merely strengthen the response to one value and weaken the response to another, however; it also increases the relevance or salience of the entire stimulus dimension. A further long-term result of experience is that different cues become functionally related or the same cue becomes effective across different conditions (for example, by being presented now to one eye, now to the other, or by being viewed from different angles of regard). Eventually, this leads to the development of functional equivalence within and across cues, expressed in equivalent responses ("recognition"), in spite of changes in conditions of presentation and the specific receptor surfaces that are stimulated.

Ganz's conceptual approach developed out of research on animals raised under conditions of highly restricted visual input, which, strictly speaking, are outside the population that we are most concerned with here. Nevertheless, I have discussed his position in some detail because it very clearly illustrates the complexities that are encountered in any systematic effort to tease out the subtle interplay between experience and innate organization in the development of schemata by advanced organisms. As far as functional outcomes are concerned, the interpenetration of "nature" and "nurture" is, for all practical purposes, complete. Learning plays an essential part in development. But most learning occurs within the framework of existing schemata. It receives guidance from these structures and is a primary agent in their transformation from phylogenetic "plans" into adaptive functional systems. According to this view, one might speculate that the learning process evolved as handmaiden of the instincts and that, even in the most advanced organisms (by which we mean ourselves, of course), it has not yet achieved full emancipation from this ancient position of subservience.

Much of the evidence that has led me to this view has come out of deprivation experiments with nonhuman primates. Some of the deficits point rather clearly to inadequate opportunities for specific forms of learning in the rearing environment. Such deficits fall into two broad classes: defective integration of sensorimotor patterns and deficiencies in signal functions. Examples of both types of deficit can be found in the social behavior of deprived animals.

The sexual performance of socially deprived male rhesus monkeys suggests a failure in sensorimotor integration.[28] Most of the components of the complete act—penile erection, clasping with the hands, clasping with the feet, thrusting—are present, but they are so poorly integrated that intromission is all but impossible. Clearly, the rearing environment has failed to provide the opportunity to form these components into an effective pattern. Accordingly, given a suitable environmental structure for rehearsal and appropriate instigation (and both conditions might be met with a suitably designed inanimate model), one would anticipate that the laboratory-reared animal would show a gradual progression toward the smoothly coordinated pattern seen in normally socialized adult males. Inasmuch as the normal pattern is not present in deprived males, it seems reasonable to infer that learning is a factor in its development. Insofar as the components of the normal pattern are present even in the socially deprived male, tend to occur together, and, under appropriate circumstances, eventually become fused into an integrated species-typical pattern, the presence of some guiding structure seems to be implied—a "template" that must be standard equipment for the rhesus macaque but remains functionally incomplete unless appropriate input is provided.

Deficiencies in signal functions can be inferred from the observation that isolation-reared monkeys do not show consistent and appropriate responses to the distinctive postures, gestures, facial expressions, and sounds that serve as the vehicles of communication in natural primate societies.[27,34] Again, however, such learning seems to operate within the context of pre-existing structures. Some configurations of stimuli and some sequences of stimulation possess primitive functions that apparently precede specific experience. These stimulus patterns are similar to the innate releasing mechanisms (IRM's) described by the ethologists, but there is no fixed relationship between stimulus and response as implied by the IRM concept. For example, young laboratory-reared rhesus monkeys display stereotyped affective-social responses toward inanimate objects that they have never before encountered. The frequency of these responses increases as the objects become more animal-like, or more representational. This effect was obtained in infants as young as 1 month, housed individually from birth in a standard nursery environment.[2]

The response to the optical stimulus of "looming" is another example of a primitive schema. Looming, created by a sudden increase in the size of circular stimulus, causes persistent fear responses in laboratory-reared infant monkeys (as well as adults).[40] The rapid approach of a solid body (which was simulated by the experimental procedure) is an important ecologic sign for the free-ranging monkey, typically associated with a potential hazard, such as a predator or an aggressive member of one's own group. At the same time, however, it is easy to see how the adaptiveness of the looming response would be improved if other input features were also taken into account. Looming can mean the approach of a hungry lion, a docile bullock, or an adult male coming to the rescue. An animal that can distinguish these possibilities and act accordingly clearly appears to be in a better position to survive than an animal that cannot. It is in this process of differentiation and evaluation of environmental contingencies that learning can be seen to play one of its most important parts.

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DISCUSSION

DR. ALPERN: As I recall from Harlow's data on rhesus monkeys, there is no evidence of any learning deficits in isolation-reared animals. But you do find it with the chimpanzee?

DR. MASON: Yes. There is no evidence of such an effect in the data on monkeys at the University of Wisconsin. I think Harlow points out, however, that they took great pains to ensure that the animals were adapted to the test situation before formal testing was started. He also indicated that the isolation-reared animals were slower to adjust to the testing procedure and that not all the animals were able to adapt. This means that considerable training and some selection occurred before formal testing began. The animals that completed the preliminary training showed apparently normal intellectual performance.

DR. INGRAM: I suggest that the data on Harlow's rhesus monkeys are rather dubious in any event. Broadbent has recently criticized the methodology of the Harlow experiments, and has suggested that many of the conclusions about cognitive development are unjustified. Broadbent in particular feels very strongly that cognitive development is impaired, but this has not been shown yet.

DR. MASON: I certainly would expect to find a variety of intellectual deficits in deprived monkeys or apes. What I am saying is that, if I did not expect it, then I would be accepting a kind of a faulty psychology, in which descriptively separate endpoints or activities are viewed as representing separate mechanisms, like so many different watertight compartments, with the animal developing intellectual abilities here, and social behavior there, and emotional behavior somewhere else. I do not believe that psychologic development proceeds in that fashion. Actually, learning, broadly defined, enters into all aspects of behavioral development, just as unlearned patterns do. Normal social development requires learning. It is one aspect of intellectual development, and all the adaptive tasks that an animal must perform provide the occasion and the necessity for the development of "intellectual" skills.

DR. GAARDER: I wonder if in some ways your position on dyslexia would tend to support the idea that dyslexia could be, not so much a derivative of many of the things that we have discussed, but rather the result of a somewhat higher process that we do not yet have adequate tools to study. This higher process has to do with the sequencing of a chain of interactions between an organism and its environment, both the nonliving parts of the environment and the human beings that are in the environment.

DR. MASON: I think a large element has been left out of our thinking about behavioral development: the ecologic element. Psychologists (ethologists, too, for that matter) lack a precise language for dealing with the microecology. Ultimately, we will have to treat the environment, not as a vast aggregate of stimuli, but in terms of its structure, because it is this structure that is reflected in the evolution of behavior. All deprivation studies deal with the impact of some drastic environmental events on ongoing, adaptive, and highly organized biologic systems. We need to develop as much concern and skill for dealing with those events and the systems that they act on as we have for dealing with behavioral outcomes. The tendency at the moment is to select the outcome—the performance—and try to infer the antecedents from that. In the long run, we must do better. We may have to start with some very messy antecedent conditions and try to get some idea of their specific relationship to the performance deficits that are produced. Eventually, we may develop sufficient sophistication to deal with the kind of higher-order process referred to by Dr. Gaarder. And the preparation of my paper gave me the opportunity to take a small step in that direction. I do not think we will ever get very close to dyslexia by using monkeys, but I do think we may get some general notion from them as to how such higher-order information-processing systems function and what they require from the environment in order to develop effectively.

JEROME KAGAN

Continuity in Cognitive Development During the First Year of Life

Each generation of psychologists seems to discover a fresh set of phenomena and a sparkling new object of study. The introspectively trained adult was the pet of the academic before and around the beginning of this century, and the contents of consciousness the favored theme. The exploring albino rat captured the stage from the brooding adult when the academy decided that public responses and easily induced drives were more critical than feelings, sensations, or thoughts. One of the stars of this decade is the human infant, and the theme turns once again to those mental processes called "cognitive structure."

The ease with which loyalties to subjects or subject matters are broken is to be expected in a discipline as young as psychology. The behavioral sciences are in the important but necessarily frustrating period of defining the phenomena they wish to pursue in depth and the concepts they must quantify and place in propositional form. The behavior of psychologists during the last three quarters of a century is not unlike that of a person who has a blanket thrown over him as he stands in a large room containing a variety of interesting objects. His task is to decide, without removing the opaque cover, what objects are in the room. It would be proper strategy to walk until he hits something, put out a hand, and explore the object to see whether a quick determination is possible. If a few minutes of exploration proves fruitless, no worry; there are many other objects in the room that can be explored. Our temporarily blinded subject would pass from object to object, exploring quickly, hoping for a clue that might define one object permanently but always prepared to pass on if an initial probe does not prove rewarding.

This horizontal progress is a fair description of psychology's first hundred years. We probe the subject–an idea or phenomenon–until we become frustrated conceptually or methodologically; then we look for another love object. We turned from introspection because we were unable to solve the subjectivity problem. We grew bored with rats and mazes because we could not solve the reinforcement problem and were unable to explain human thought by turn sequences in alleys. We abandoned personality study because we failed to develop sensitive procedures to measure motives, anxieties, or conflicts. And now we affirm the child.

THE CONCERN WITH CHILDHOOD AND INFANCY

Interest in the young child, especially the infant, draws its force from divergent sources. The commitment to historical explanation, which is basic to American psychology, has always been a guide to domestic empiricism. A large proportion of studies on rats, cats, mice, and monkeys have posed developmental questions. Harlow and his colleagues[18,19] asked about the later effects of infant experience with terry-cloth mothers. Denenberg[7] and Levine[28] inquired into the effect of mild handling of week-old mice on adult behavior in an open field. The historical bias, which is not shared by all psychology groups, takes its strength from a strong belief in the persistence of well-learned habits (an heir of Hullian behaviorism) and an optimism that affirms that what was done can be either undone in the same organism or done differently in the next generation.

The influence of psychoanalytic theory, although waning rapidly, also directed our interest to the child. As the major theory of personality development, it made strong statements about the long-term effects of family experience during the first 5 years.

Finally, the unprecedented excitement about educational progress in the lower-class child, an excitement that has always been in the background, has suddenly come into focus and produced a heady dose of investigations into the mental processes of young children. There is agreement that one can preview the awesome class differences in school-age children as early as the age of 3, but we do not know the temporal patterning of events during the prenatal months and the first 36 postnatal months that lead to this variation.

Although scholarly concern with childhood has a broad foundation, the more limited interest in the infant required a special catalyst, and it was provided by Robert Fantz's demonstration that one could tell with remarkably simple methods what a baby was looking at and perhaps what he preferred to look at.[9,10] To everyone's surprise, the very young infant was not perceptually innocent. He stared longer at checkerboards and bull's-eyes than at homogeneous gray patterns[9] and more at moving lights than at stationary ones.[17] As early as the age of 4 months, he looked longer at a picture of a human face than at nonsense designs.[31,32] Moreover, the infant always seems to be seeking variety.

PERCEPTUAL LEARNING

Does the infant learn anything when he looks at an object, and if he does, what does he learn? These simple questions have generated considerable controversy because of an unstated faith that the infant was cognitively different from the adult. If an adult were to scan a photograph of Lake Atitlán for the first time he would learn and retain the fact that it has a very irregular perimeter. The adult would not have to feel the photo, swim in the lake, or draw its outline on paper. At a more conceptual level, if an adult is told that an operon is a concept used by molecular geneticists to explain suppression of structural genes, he has only to listen to this statement to learn the meaning of this concept. At a behavioral level, Bandura and Menlove[2] have shown that a 3-year-old child who is afraid of dogs will approach and play with them after watching a short series of films showing children playing with dogs. These examples are sufficient to warrant faith in the commonsense proposition that an older child or adult can learn a perceptual structure, a conceptual unit, or changes in the hierarchy of overt behaviors by merely looking or listening. But is this true of infants?

Investigators of mental growth in the human infant before the 1960's were prejudiced against this view. Tests constructed to assess infant intelligence emphasized motor accomplishments–age of sitting, walking, hand–eye coordination, removal of a napkin from a toy, prehension of a button. The implicit assumption was that the child's level of intellectual development covaried with his precocity in sensorimotor acquisitions. This reasonable belief was supported by the major cognitive theorists of this century. Jean Piaget[36] stated explicitly that the infant's first mental structures (schemata) are "interiorizations of overt actions," a view resembling Watson's belief that thought was derived from subvocal speech. Gesell, Piaget, and Watson probably arrived at their faith independently; none was especially devoted to the others' writing during the first half of the century, and there has been minimal contact among the loyal disciples of each of the three leaders. Such remarkable consistency is often a reliable sign of validity.

The reasons for this commitment to the value of actions in mental growth are probably different. The early behaviorists were in a methodologic rebellion, and it was important for its vitality to forbid ideologic deviance. No concepts were permissible that could not be measured publicly. The intelligence testers were probably moved by pragmatic considerations and Gesell's clearly described procedures.[15] There was no way to assess what the infant was thinking, but there were ways of measuring what he was doing, and the American attraction to reasonable solutions makes it easy to understand why they focused on overt behaviors. It is difficult to understand Piaget's affinity for this materialistic view of infant intelligence. Piagetian theory is profoundly cognitive, and his epistemologic position would lead one to expect a more mentalistic flavor to infant psychology.[35,36] Perhaps his autobiography will solve that puzzle.

Existing empiric data suggest that an infant may acquire a mental representation of an event by only looking or listening. An infant who has habituated to a repeated presentation of the same, initially novel, stimulus shows a dramatic change in fixation time when presented with a transformation of the stimulus. This phenomenon can be demonstrated in studies using short-term habituation (McCall and Kagan, unpublished data) or exposure of an infant to a stimulus over a period of several weeks.[31,32] These data imply that the infant, as well as the older child, establishes cognitive structures as a result of exposure to an event.

CONTINUITY IN MENTAL DEVELOPMENT

The mechanisms of establishment of cognitive structures are separate from the issue of continuity in level of cognitive development. Do infants who at some time show precocious acquisition of a class of structures remain precocious? That is the question the early infant testers wished to answer. Their efforts were ambiguous, because they evaluated behavioral accomplishments in the infant and cognitive accomplishments in the older child. Although demonstration of stability or continuity is one prerequisite for a theory of intellectual development, stability is necessarily ambiguous as to cause. Suppose, as our data and those of others indicate, that there is a high correlation between reliable indexes of vocalization in 4-month-old girls and verbal ability at the age of 2 years. One might view this correlation as proof of a constitutional basis of intelligence. The argument would state formally that the same central intellective process caused the frequent vocalizing at 4 months and the high verbal score at 2 years. This phenomenon might be called "endogenous continuity" (i.e., a particular internal process remains stable over time). However, it is possible that an environmental force, such as a mother's reciprocal interaction with her child, produced both the frequent vocalization in infancy and a rich vocabulary and strong motivation for cognitive mastery in the older child. The latter forces produced the high verbal score. There is no necessary causal relationship between the vocalization at 4 months and the later verbal proficiency. It is possible that each is an independent correlate of different sets of maternal behaviors and that the stability was in the mother's behavior. This phenomenon might be called "exogenous continuity."

Value of Continuity Studies

The issue of continuity is relevant to three themes in developmental psychology. First, knowledge of responses that show stability facilitates prediction of future behaviors and early diagnosis of psychologic syndromes that are harmful and should be treated, as well as of socially valued syndromes that should be protected and encouraged.

Second, continuity studies contribute to the validation of major theoretical positions. Knowledge of continuity in behavior allows tests of, for example, whether frustration of oral needs in infancy leads to depression in adulthood and whether partial reinforcement of aggression in childhood produces greater resistance to extinction of aggressive behavior during later childhood. Validation of these theoretical predictions, which requires studies of behavior over time, is necessary to evaluate the power of the theoretical schemes.

The third rationale for continuity studies is the one that will be discussed here. Studies of continuity facilitate the understanding of the meaning of responses at particular ages. Contemporary psychology does not understand the meaning of some of its simplest phenomena, such as the smile at 4 months, separation anxiety at 10 months, attempts at task mastery at 1 year, and the early attainment of what Piaget calls the "object concept." It is not clear what covert processes each of these public phenomena reflects. Study of the predictive consequences of selected responses may clarify the significance of the act.

Indexes of Mental Development

A central methodologic problem common to many investigations of mental development is lack of agreement on the behavior patterns that provide a faithful picture of the infant's mental development. The classic conceptual stance assumed that selected motor and sensorimotor responses mirrored the level or rate of cognitive development. These motor patterns are disrupted by brain-stem damage, and anomalies in their development are used as diagnostic signs of lack of integrity of the central nervous system. But the rate of development of these complex motor responses may not be the most sensitive index of the infant's cognitive development ("cognitive" refers to perceptual structures, language, and problem-solving skills). Among infants with no central nervous system damage, there appears to be a minimal relation between age of appearance or quality of these sensorimotor patterns during the first year and standard indexes of language and problem-solving ability during the preschool years.[3,8]

However, responses that index differential attentiveness to selected events may reflect differential acquisition of cognitive structures related to those events, and they may provide evidence of intra-individual continuity of cognitive dimensions from infancy to early childhood. Duration of orientation toward a visual stimulus ("fixation time") and vocalization during stimulus presentation are two reasonable indexes of an infant's degree of attention to an external event.

FIXATION TIME

The intuitively obvious index of visual attention is the duration of study of an event. Like most acts, fixation time has multiple determinants, and the power of each seems to change with age. Physical contrast, movement, discrepancy from an acquired schema, and density of hypotheses activated to explain a discrepancy all exert some control on fixation time during the first 30 months of life.

Movement and Contour

Ontogenetically, the first determinant of orientation toward a visual event is a high rate of change in the stimulus itself, typically produced by movement or contour contrast. The infant has an unlearned disposition to attend to events that possess a high rate of change in their physical characteristics. Newborn infants are dramatically more attentive to moving lights than to static ones and to designs with a high degree of black–white contour than to events with minimal contour contrast.[11,17,38] These induced generalizations from molar behavior concur with neurophysiologic studies of ganglion potentials in vertebrate retinas. Some ganglion cells respond to onset of illumination, others to offset, and still others to both. A stimulus moving across a visual field stimulates sets of cells for a short period, creating onset and offset discharge patterns. Contour edges function as onset stimuli, whereas solid patterns do not; and the change in stimulation created by a sharp contour edge or movement elicits specialized firing patterns that can serve as the basis for sustained fixation.[25,26]

Discrepancy

The initial disposition to maintain long fixations on visual stimuli with movement and contour eventually competes with a second, acquired determinant: the degree of discrepancy between an event and the child's acquired schema for that event. Stimuli that deviate moderately from an existing schema release longer fixations than either completely familiar events or completely novel events. Empiric support for the role of schema discrepancy on attention is suggestive, rather than definitive. At 4 months of age, achromatic illustrations of male faces elicit fixation times twice as long as those elicited by random shapes of varying numbers of turns that contain extreme novelty and greater contour contrast than the faces.[31,32] Haaf and Bell[16] have shown that 3-month-olds study a regular schematic face longer than one that has the same facial components disarranged. Moreover, the infants study partly disarranged schematic faces longer than completely disarranged ones. The stimuli that were similar to faces elicited longer fixations than equally complex stimuli with minimal resemblance to faces. Final support for the discrepancy hypothesis comes from a study in which 3-month-old infants were exposed to a novel three-dimensional stimulus at home for a month and then shown the same stimulus and three transformations of it at 4 months of age. Control children viewed all four stimuli for the first time at 4 months. The experimental infants showed shorter fixation times to all four stimuli than the controls. The discrepancy effect emerged for girls, who showed the longest fixation times to the transformations of the standard they viewed at home.[31,32] Although the definitive study demonstrating a curvilinear relationship between fixation time and discrepancy from an acquired schema is still to be done, existing data are persuasive of the usefulness of this idea. At the least, they suggest that the degree of familiarity, independent of movement and contour qualities, does exert a powerful influence on fixation time.

Density of Associations

A third determinant of fixation time begins its growth during the second half of the first year and involves the density or richness of the symbolic associations activated by the child to assimilate experience. The richer the nest of hypotheses available and activated, the longer the fixation.

High rate of change, discrepancy, and activation of hypotheses may be additive in their total effect on fixation time. The combined effect of discrepancy and hypotheses is illustrated in a comparative study of 1-, 2-, and 3-year-old children from two cultures. Finley[12] showed culturally appropriate illustrations of male faces and forms to Cambridge, Massachusetts, middle-class children and Mayan peasant children from Yucatan. Fixation time increased linearly with age, for both groups, as predicted from the principle of increased density of hypotheses. But the largest increases in fixation time occurred in response to a disarranged face or form, which is a discrepancy stimulus, rather than to the regular face or form. The hypothesis vector complemented the discrepancy vector to produce a significant age–stimulus effect on fixation time.[12]

The changing pattern of influences on fixation time should result in different patterns of continuity over time, and, as we shall see, that expectation has been verified.

VOCALIZATION

The determinants of infant vocalization are much less clear. There is a dramatic increase in positive vocalization at about 10–12 weeks of age, accompanied by an equally salient decrease in fretting and irritability.[27] Infants often babble when they study an interesting visual stimulus or hear selected auditory events, and it is reasonable to suggest that these vocalizations reflect the excitement that accompanies focused attention. Thus, one early determinant of vocalization may be similar to the discrepancy principle that states that partially novel events engage the infant's attention and elicit vocalization. Around 1 year of age, it appears that the hypothesis vector may also control vocalization: Finley found linear increases in vocalization in response to human faces and forms across the period from 1 to 3 years of age in both the American and the Mayan children.

This report is conçerned primarily with the intra-individual stability of fixation time and vocalization in response to representations of human faces during the first year of life. It is believed that the pattern of this stability provides information on continuity of acquired cognitive structures associated with human faces.

A subordinate theme is the possibility that vocalization during the early months has different meanings for the two sexes. Vocalization may display a sexual dimorphism in the human infant. There has been a remarkable change in our attitude toward the etiology of human psychologic sex differences, in that we now acknowledge that these differences may not all be environmentally determined. Infrahuman studies have demonstrated sex differences in dominance, as well as prepotent reactions to stress or threat.[6,13,19] Recent reviews of the comparative literature by Hinde[20] and Marler and Hamilton[30] indicate strong support for sex differences in a variety of response dimensions in mammalian species. It is reasonable, therefore, to expect sex differences in initial response hierarchies that would lead infant boys and girls to react differently to the same stimuli or environmental intrusions.

PROCEDURES

Our total sample consisted initially of 91 boys and 89 girls; all were Caucasian, firstborn, and living with their natural mothers within a 30-mile radius of Cambridge, Massachusetts. The range of educational level of the parents of these children ran from 8 years through a graduate degree. Most of the parents of the sample children were volunteers recruited by advertisements in local newspapers. Each infant came to the laboratory with its mother when the infant was 4, 8, and 13 months old for a series of assessments.

Each 4-month-old infant was placed supine in a crib. When the infant was quiet and content, but alert, he was shown a series of 16 achromatic slides of four different human faces (Figure 1); there were four presentations of each of the four faces. After a short recess, the child was shown a series of slides of four different three-dimensional clay faces (4½ X 6½ in.) painted flesh color (Figure 2); there were four presentations of each of the four clay faces. For both episodes, the order of presentation was such that each of the four faces appeared once every four slides, and there were two different orders of presentation. Each stimulus was shown for 30 sec, with 15-sec intervals between exposures, during which the visual field was homogeneously white. The stimuli were presented 20 in. from the plane of the child's face and occupied a visual angle of about 20 deg.

Two of the variables coded during these two episodes were length of each fixation on the stimulus and duration of vocalization during stimulus presentation. These variables were coded by two independent observers watching from opposite sides of the crib, neither of whom could see the stimulus being presented to the child. Interobserver reliabilities were 0.97 for fixation time and 0.71 for duration of vocalization.

When the infant returned to the laboratory at 8 months of age, he was seated in a high chair behind a gray enclosure with his mother seated to his right and slightly behind him. A screen was 2 ft in front of the child at his eye level. The first episode was the same set of four achromatic faces that had been presented at 4 months. However, each

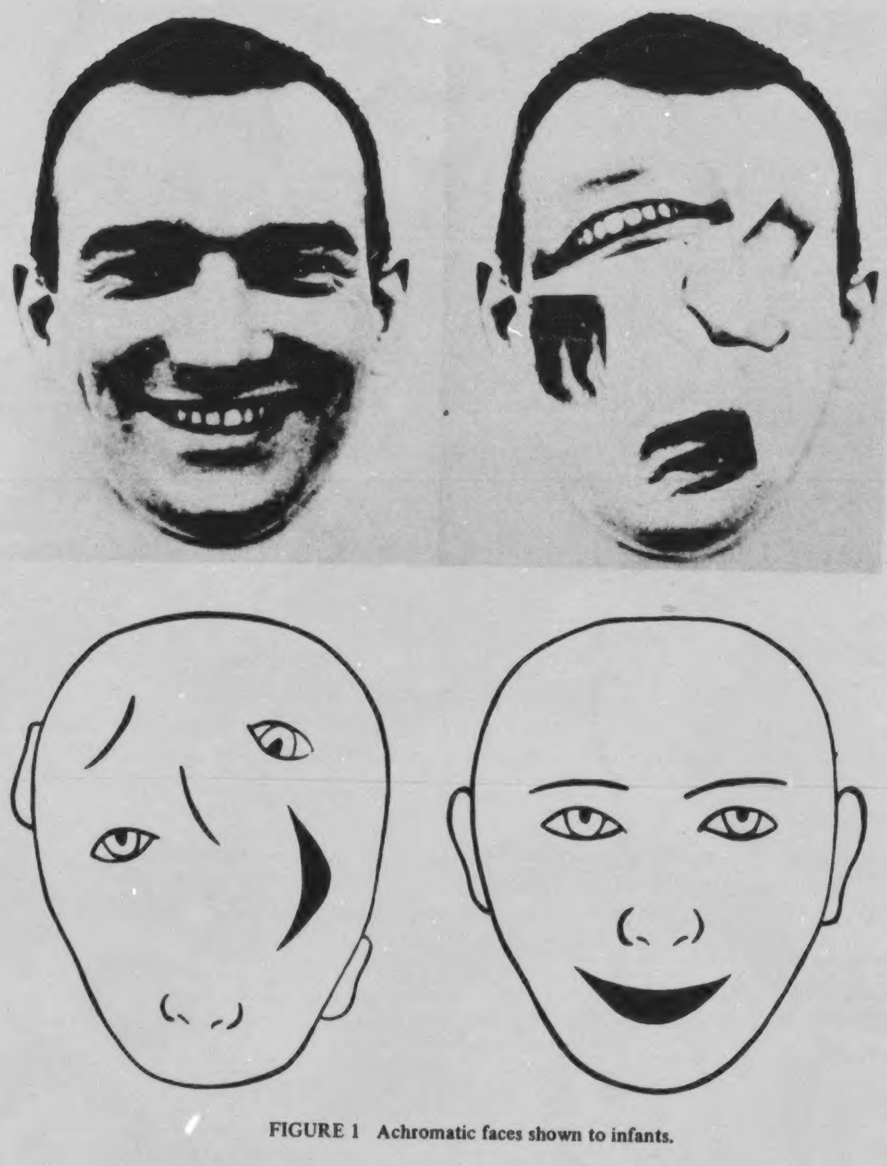

FIGURE 1 Achromatic faces shown to infants.

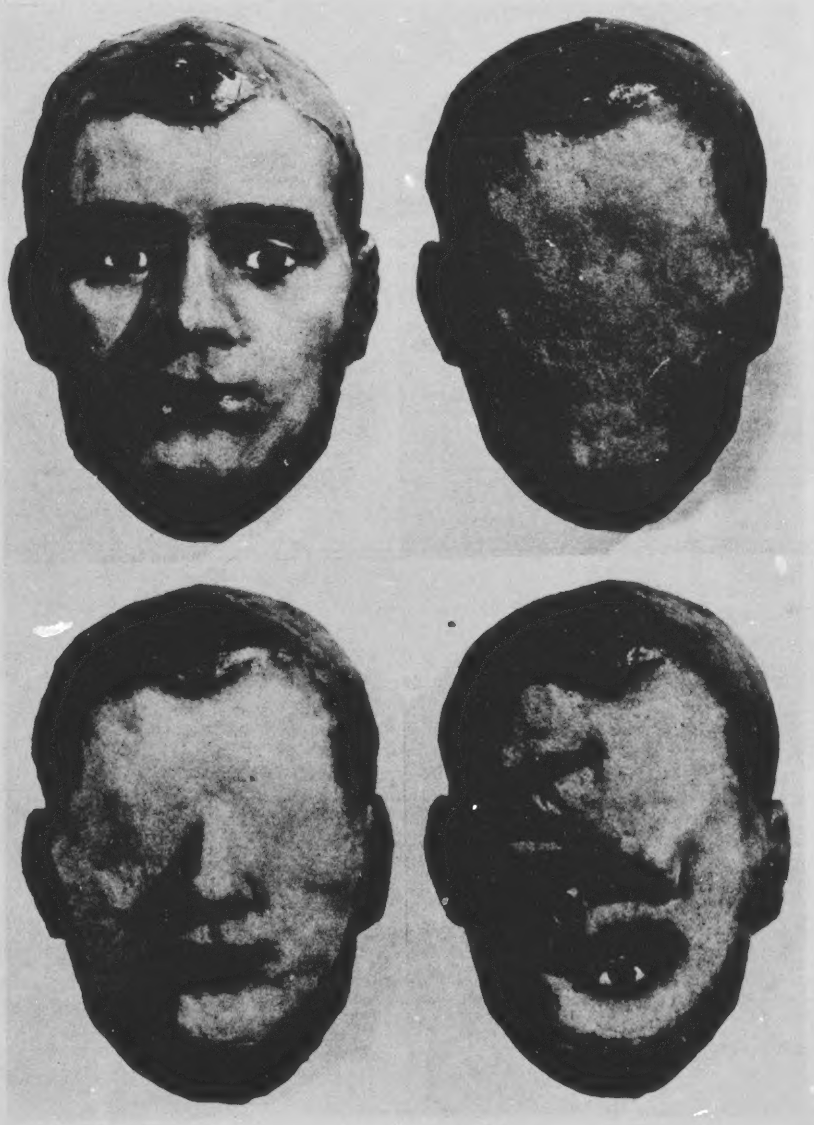

FIGURE 2 Clay faces shown to infants.

TABLE 1 Correlation Coefficients of Fixation Times from 4 to 13 Months of Age

	Predictor Variable															
	4 Months								8 Months							
	Achromatic Faces				Clay Faces				Achromatic Faces				Clay Faces			
	First Fixation		Total Fixation		First Fixation		Total Fixation		First Fixation		Total Fixation		First Fixation		Total Fixation	
Criterion Variable	Boys	Girls	Boys	Girls	Boys	Girls	Boys	Girls	Boys	Girls	Boys	Girls	Boys	Girls	Boys	Girls
8 months																
Achromatic faces																
First fixation	0.14	0.00	0.07	0.13	0.12	–0.01	0.00	0.11								
Total fixation	–0.02	0.03	–0.07	0.10	0.03	–0.20	–0.01	–0.05								
Clay faces																
First fixation	0.17	0.29[a]	0.16	0.13	0.26	0.14	0.15	0.14								
Total fixation	0.23	0.31[a]	0.13	0.12	0.22	0.05	0.18	0.03								
13 months																
Clay faces																
First fixation	0.06	–0.05	0.06	–0.08	–0.04	–0.16	–0.06	–0.01	0.20	0.26[a]	0.21	0.44[b]	0.16	0.24	0.13	0.30[a]
Total fixation	0.12	0.00	0.04	–0.04	0.00	–0.02	0.02	0.05	0.16	0.27[a]	0.24	0.41[b]	0.00	0.48[b]	0.06	0.51[b]

[a] $p < 0.05$; two tails.
[b] $p < 0.01$; two tails.

stimulus was presented for only 15 sec, with 15-sec intervals between exposures. The second episode was a tape recording of four different 20-sec recitations read by a male voice, with 10-sec silent periods between recitations. Two of the recitations contained the same set of meaningful sentences, one set read with high inflection and the other with no inflection. The other two recitations contained the same set of meaningless sentences (nonsense words), one read with high inflection and the other with no inflection. Each stimulus was presented three times in a fixed order that was the same for all the children. The third episode was the same set of clay faces that had been presented at 4 months; the procedure was identical with that used at 4 months.

Fixation time and duration of vocalization were again coded during the presentation of the faces. Vocalization time and duration of orientation toward a speaker baffle were coded during the auditory episode. The speaker baffle was 1 ft above and 45 deg to the right of the child's right eye. Many of the children oriented their heads and eyes toward the speaker baffle when the recorded voice was being presented.

Each 13-month-old infant was exposed to four episodes in the same apparatus used at 8 months. This report is concerned only with the third of these four episodes, during which the child was shown a set of five different clay faces. Four of the faces were identical with those viewed at 4 and 8 months. The fifth face contained a pair of eyes correctly placed, but no nose or mouth. Each of the five faces was presented three times in three different orders of presentation. Each face was presented for 30 sec, with 15-sec intervals between exposures. The duration of each fixation and vocalization was coded.

RESULTS

Fixation-Time Stability

Two fixation-time variables will be considered: average first fixation and average total fixation across all stimulus presentations in an episode. First fixation times averaged 8 sec at 4 months and 5 sec at 8 and 13 months. Total fixation times averaged 17 sec at 4 months and 8 sec at 8 and 13 months. There were no significant sex differences in average fixation time or variability at any of the ages. Table 1 presents the stability of first and total fixation times across the three ages expressed as the coefficients of product moment correlations; sample sizes are usually 60 boys and 60 girls.

There was minimal intra-individual stability of fixation time from 4 to 8 or from 4 to 13 months for either sex. There was moderate continuity from 8 to 13 months for girls, but not for boys. It is relevant to add that both first and total fixation times at 8 months covaried positively with the parents' educational level for girls, but not for boys. When the mean parental educational attainment was divided into four steps, analysis of variance on the total-fixation-time scores revealed a significant variance ratio (F) for girls ($F = 3.49$; 3, 75 df; $p < 0.05$ for achromatic faces; $F = 2.28$; 3, 61 df; $p = 0.09$ for clay faces). The corresponding variance ratios for boys were less than 1.0. The greater stability of fixation time for girls was accompanied by evidence that social-class membership was influencing fixation time. The favored interpretation of these data is that duration of fixation time at 4 months is primarily a function of degree of discrepancy between the stimulus faces and the child's schema of a face. The primary determinant of fixation time at 8 and 13 months, however, is the density of hypotheses activated to assimilate the faces. If middle- and upper-middle-class girls have acquired richer nests of hypotheses to faces, they should have longer fixation times. This interpretation suggests that the stability of fixation time from 8 to 13 months reflects continuity of a dimension that we can call "richness of cognitive structures associated with the human face." This continuity is analogous to the typical correlation of 0.50 between vocabulary levels at the ages of 3 and 6 years. Children who are precocious in the acquisition of hypotheses to faces at 8 months retain this precocity for the next half-year.

Vocalization-Time Stability

Mean vocalization time was highest at 4 months (average, 2 sec), lowest at 8 months (average, 1.2 sec), and intermediate at 13 months (average, 1.5 sec). Vocalization in response to the faces typically occurred while the infant was looking at the face. Vocalization during the auditory episode at 8 months typically occurred when exposure to the stimulus ended, not during it. The children were usually quiet and attentive while the voice was speaking. When it stopped, babbling began, as though the vocalizations were released by the processing of the sounds.

There were no significant sex differences in mean vocalization times or variance ratios to any episode.

Table 2 presents the product moment correlation coefficients for vocalization times across the three ages. As with fixation times, there was no stability from 4 to 8 months for either sex. However, vocalization time showed suggestive stability for girls from 4 to 13 months and moderate stability from 8 to 13 months. There was no consistency for boys between any two ages. The sex difference in stability of vocalization is not linked to social class; there was no strong relationship between vocalization and educational level of the parents at any age. Despite the absence of sex differences in duration or variability of vocalization, it is suggested that vocalization reflects processes closely related to attentiveness more faithfully in girls than boys. The girls' stability coefficients imply continuity of an attentiveness dimension in girls. Let us consider support for this statement.

The 4-month-old girls who vocalized in response to the achromatic faces displayed longer visual orientations to the speaker baffle at 8 months; this relationship did not occur in boys ($r = 0.28$, $p < 0.05$ for girls; $r = -0.10$ for boys; r is the correlation coefficient). This sex difference in the predictive significance of vocalization is seen in bolder relief when the analysis is restricted to infants with long fixation times (i.e., above the median on first fixation at 4 months). This group of long-fixation 4-month-old infants was divided at the median for the vocalization score into those who vocalized 1 sec or more and those who vocalized less than 1 sec in response to the achromatic faces. The high-vocalizing 4-month-old girls oriented to the speaker more often than the low-vocalizing girls (8 versus 2 sec; $p < 0.05$). The differences for boys were the reverse of those for girls. High-vocalizing boys oriented less often toward the speaker than low-vocalizing boys (4 versus 8 sec; $p < 0.05$). If orientation toward the source of the human voice at 8 months is regarded as a partial index of a tendency to attend to interesting auditory stimuli, this disposition is predicted by frequent vocalization in the 4-month-old girl, but by infrequent vocalization in the 4-month-old boy.

A second source of support for the hypothesis that vocalization is a more faithful index of attentional processes in girls than in boys comes from the covariation between duration of first fixation and vocalization at 4 months. The distribution of first fixation times to each stimulus

TABLE 2 Correlation Coefficients of Vocalization Times from 4 to 13 Months of Age

	Predictor Variable									
	4 Months				8 Months					
	Achromatic Faces		Clay Faces		Achromatic Faces		Clay Faces		Auditory Episode	
	Vocalization to Stimulus								Vocalization Following Termination of Stimulus	
Criterion Variable	Boys	Girls	Boys	Girls	Boys	Girls	Boys	Girls	Boys	Girls
8 Months										
Achromatic faces										
Vocalization	0.02	0.00	0.18	–0.04						
Clay faces										
Vocalization	0.00	0.01	0.17	0.03						
Auditory episode										
Vocalization following termination	0.12	0.01	0.10	0.06						
13 Months										
Clay faces										
Vocalization	–0.07	0.01	–0.03	0.33[a]	–0.02	0.34[b]	–0.14	0.46[b]	0.08	0.46[b]

[a] $p \leq 0.05$; two tails.
[b] $p \leq 0.01$; two tails.

was split into thirds, and the distribution of vocalization into halves (i.e., equal to or greater than 1 sec versus less than 1 sec) for each trial. The probability of vocalizing 1 sec or more increased linearly with length of first fixation for girls, but was independent of fixation time for boys.

The pattern of intercorrelations at 8 months furnishes additional support for the attentional significance of girls' vocalization. Boys and girls were equally likely to vocalize at the end of each of the auditory recitations at 8 months. But the vocalization was positively associated with magnitude of cardiac deceleration (one index of orientation to an event) on the preceding trial for girls, but not for boys. For example, the correlation between vocalization following the high-meaning–high-inflection stimulus and magnitude of cardiac deceleration during the presentation of the stimulus was 0.36 ($p < 0.01$) for girls and only 0.01 for boys. The girls who vocalized after voice stimulation showed the largest cardiac decelerations in response to the voice. Inasmuch as cardiac deceleration is one reasonable index of quieting in the service of attention,[29, 31, 32] the girls' babbling seems more clearly in the service of attentional processes than the babbling of boys.

A fourth line of evidence is based on the girls' differential vocalization in response to the faces. In the first presentation of the faces, the girls vocalized more in response to the most regular representation (photo regular) than to the schematic representation (schematic regular), whereas the boys' vocalizations were equivalent (2.2 versus 1.5 sec for girls; 2.5 versus 2.4 sec for boys). Similarly, the girls vocalized twice as long in response to the regular clay face as to the scrambled clay face (3.7 versus 1.9 sec); the boys' scores were almost equal (2.6 versus 2.8 sec).

This differential vocalization is supported by an independent investigation of 32 girls and 32 boys, 6 months old, who viewed achromatic faces and geometric forms in the same series. The girls vocalized significantly more in response to the faces than to the forms; the boys' vocalizations were equal for both classes of stimuli (Lewis and Kagan, unpublished).

The special link between early vocalization and indexes of level of cognitive development in girls is supported by the research of others. Infant girls 6–12 months old with high scores on a vocalization index derived from the Bayley Infant Intelligence Scale had high Stanford-Binet IQ scores during the period from 6 to 26 years of age (correlations ranged from 0.40 to 0.60 during adolescence and early adulthood). There was no relationship between the boys' vocalization scores and IQ during the later years.[5] Only the girls' early babbling previewed a future level of cognitive development.

A final source of evidence is a longitudinal study in London in which 41 boys and 35 girls were seen at the ages of 6 and 18 months and 2, 3, 4, 5, and 8 years.[33] At 6 and 18 months, each child was assigned a speech quotient from the Griffith's Infant Scale, which assessed spontaneous babbling at 6 months and use of words at 18 months. Although there were no sex differences in mean speech quotient at 6 or 18 months, the speech quotient was more stable from 6 to 18 months for girls than for boys ($r = 0.51$ for girls versus 0.15 for boys). Furthermore, the speech quotient at 6 months predicted vocabulary level at 3 years for girls ($r = 0.52, p < 0.01$) but not for boys ($r = -0.01$).

SUMMARY

Despite different procedures, populations, and *a priori* hypotheses, the data from Cambridge, Yellow Springs, Berkeley, and London are remarkably concordant in suggesting that babbling in response to human faces or voices is more stable for girls than for boys during the first year and a better predictor of future indexes of level of cognitive development for girls than for boys. There are at least two possible interpretations of this empiric generalization. It can be argued from a purely environmental position that mothers who are motivated to accelerate their daughters' mental development are likely to spend a lot of time in face-to-face vocalization with them—more time than they would with sons, and more time than would mothers who are not overly concerned with their daughters' rate of development. A mother's face-to-face vocalization should lead to increased levels of babbling in her daughter.[37] However, such a mother would also be expected to continue to stimulate her daughter and would probably teach her words early and encourage the development of other skills that are measured on intelligence tests. The predictive link between early babbling and later cognitive abilities would be a function of the continuity of a mother's acceleration of her daughter's development. The absence of this predictive link between infant vocalization and cognitive development in the boy would require the assumption that accelerating mothers do not preferentially engage in face-to-face vocalization with their sons. Preliminary data support this assumption. Observations of mother–infant interactions in the home reveal that well-educated mothers engage in more distinctive face-to-face vocalization with their daughters than do less-educated mothers, whereas there is no comparable difference among the mothers of sons.[24] Moreover, Moss[34] has reported that middle-class mothers are more likely to imitate the vocalization of their 3-month-old daughters than that of their sons.

A second interpretation has a more biologic flavor and holds that there is a basic sex difference in neuromotor organization. It is possible that vocalization is more prepotent for girls than for boys as a behavioral reaction to the arousal occasioned by the processing of interesting stimuli. This more speculative position finds some support in natural observations in primates. Among the langur of northern India, for example, squeals and screams are observed more frequently among females than among males.[21] More important, however, is the generally accepted premise that closely related strains or the sexes within a strain can differ in their typical reactions to states of arousal.[6,39] This generalization might hold for male and female infants with respect to early vocalization.

Aside from possible sex differences in the significance of vocalization, it is to be noted that both vocalization and fixation time displayed better stability from 8 to 13 months among girls than among boys. Indeed, there was minimal continuity for the boys. This finding is paralleled by data on older children, indicating greater long-term stability for girls on a variety of cognitive dimensions, including intelligence quotients[4] and decision times in problem situations.[23] Although it is possible to explain the older girls' greater continuity as a result of socialization experiences, there may be some biologic basis to this sexual dimorphism in cognitive functioning. Girls manifest greater stability than boys on a variety of physical-growth variables, including onset of ossification centers,[14] and Acheson[1] has concluded that "in almost every respect the physical development of the female is more stable than that of the male"(p. 497). The biologic processes that mediate girls' more stable physical growth may also be partly responsible for their more impressive stability in psychologic dimensions during infancy, as well as later childhood. At the least, the data question the popular myth of female unpredictability.

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DISCUSSION

DR. KAGAN: I want to add to the first empiric generalization summarized in my paper, dealing with the changing control of fixation time in the young infant. The second generalization, which I hold with a little less confidence than I do the first, is an idiographic issue, rather than a nomothetic one.

We noticed both at the Fels Research Institute and in Cambridge a covariation between the style of the child's habituation and his pattern of play, and this story takes us to age 7 and reading ability.

We put children at three ages in a free-play situation. (These are the same longitudinal studies from which we gathered the previous data.) At 8 and 13 months, a child is placed in a room about 11 ft square (the mother is asked to sit in a chair and not say anything), is given age-appropriate toys, and is allowed to play for about a quarter of an hour. We code the rate at which the child changes his attentional involvement.

Some children pick up a toy, play with it for 10 sec, drop it, pick up another toy, drop it in 5 sec, and so on. Another child picks up a toy and stays with it for a minute. He does not do anything more creative with the toy—he does the same thing that the other child does; but he does not turn his attention from it so quickly.

There appear to be stable individual differences in the rate at which children change attentional involvement. Moreover, there is a correlation for boys, but not for girls, between rate of habituation to visual stimuli at 4 months and pattern of play. Specifically, rapid habituators at 4 months show a fast tempo of play at 8 months, with many act changes in their 15-min free-play period, whereas children who are slow habituators at 4 months show long periods of attentional involvement in play.

At 27 months of age, the children play in a larger room, decorated as a living room. The mother sits on a couch reading a magazine, and a new set of toys is introduced. We record the duration of each attentional involvement during the half-hour session. Children 27 months old tend to play with a toy for 30 sec before they turn their attention from it, but there are dramatic individual differences. Some children have a modal attentional involvement of 20 sec with no act lasting longer than 100 sec. Some have a mode of 40 sec with many acts that last 2 or 3 min.

We then asked whether there was a relationship between tempo of play at 8 months and at 27 months of age. There was for boys, if one looked at the extremes. That is, fast-tempo boys at 8 months tend to be fast-tempo boys at 27 months.

We believe that tempo of play, like any behavior, is determined by multiple factors. Obviously, the richer the set of hypotheses the child has for a particular object, the longer he will play with it. But, as a working idea, we suggest that there may be a biologic variable that exerts some influence on the tendency for a child to develop into a fast- or slow-tempo person. When we compared nine boys who were slow-tempo versus nine who were fast-tempo at 27 months, the slow-tempo boys had slightly shallower habituation curves at 4, 8, and 13 months, with the difference greatest at 4 months. The differential habituation curves to the faces at 4 months suggest that there is a tendency for fast-tempo boys to have been rapid habituators as infants. We should remember that we are talking about the top and bottom 10%, and not the whole population.

We have worked for several years on a psychologic dimension in school-age children that we call "reflection–impulsivity." This tendency is assessed in a perceptual match-to-standard test in which the child is shown a picture and must pick from a set of six or eight variants the one that matches the standard. Some children offer their first hypothesis in 4 sec, and they are often wrong. Some wait 30 sec and are usually correct. The former are called "impulsive," the latter, "reflective." The correlation between errors and response time on this task is typically negative.

The tendency to be reflective or impulsive generalizes across tests and tasks. We also found that this tendency tends to be stable over short (12 weeks) or long (1–2 years) periods.

What makes a child reflective or impulsive? Most of the variance, we believe, is due to psychodynamic factors related to anxiety over error. If a child is anxious over making a mistake, he is likely to be reflective. If he is not, he is likely to be impulsive. But when we look at extreme children and match them on social class and intelligence, I am persuaded that a few impulsive children would find it difficult to be reflective, and a few of the reflective children would find it difficult to be impulsive. We suggest that fast-habituating, fast-tempo infants have a slightly higher probability of ending up impulsives, rather than reflectives, as a result of biologic variables.

We believe, moreover, that impulsive children are more likely to make errors in school tasks when reflection would be an advantage (Child Develop. 36:609–628, 1965). We selected children who were either reflective or impulsive, but who were of equal intelligence, and gave them a test to see the kinds of reading errors they would make. A child was given a card on which were printed "moon," "boom," "soon," and "room." The examiner then said one of the words and asked the child to point to it. Impulsive children made many more errors than reflective children. A year later, we asked the same children to read a paragraph of prose. The results will not be surprising: the impulsive children made many more errors, adding suffixes and usually missing initial graphemes (saying "trick" for "truck" or "wagon" for "witch"). These errors occurred more commonly for the impulsive than for the reflective children. Only one type of error was more frequent for the reflective children. They more frequently corrected a mistake spontaneously. The reflective child might say: "The dog ran—no, no—the cat ran."

Some evidence that tempo of play has a biologic component comes from a study of twins done by Christine Reppucci. She saw monozygotic and dizygotic twins in the free-play situation described earlier and also had them look at visual stimuli. The results suggested heritability, for this population, for tempo of play at 8 months and rate of habituation.

DR. CAVANAGH: Do your data permit you to say anything about the fluency of these children, their use of language?

DR. KAGAN: We thought that there would be a correlation between language fluency and impulsiveness, and William Ward of the Educational Testing Service ran a study (unpublished) of the fluency of verbal resources or fluency of ideas. We were hoping that the impulsive children would have greater fluency, but the correlation was low and nonsignificant.

DR. GUNDERSON: Did you find any understanding about language acquisition?

DR. KAGAN: The correlation between verbal resources and impulsivity is 0.15 for boys and insignificant, and about 0.30 for girls. In general, reflectivity is correlated with verbal IQ. Others have found that verbal fluency is more highly correlated with verbal IQ in girls than in boys.

DR. LINDSLEY: Do you associate reflectivity with perseverance, in the sense that a person stays with a thing longer? There is a two- or three-pronged aspect to this question. A study done several years ago on activities that were started and then interrupted tried to find out whether subjects would resume the same activity after the interruption. There was some evidence of perseverance, and I am sure you have seen that in animal studies when you interrupted the system through the cortex.

In relation to active children and what you called the "fast-tempo" group: All of us tend to walk fast or slow, and the temporal factors characteristic of a person may be biologically inherent or acquired. I am interested in whether reflectivity had an aspect of perseverance or long-term attention, and whether these are advantageous features. It seems to me that we must have people who can attend to what they are working on long enough for information exchange to take place, but who need not hang on so long that they cannot break off and pay attention to something else.

DR. KAGAN: When a school-age child becomes involved in a task for which there is a correct or an incorrect solution, he reflects for a variable period. It seems to me that, although an impulsive child does not care about failure, a reflective child does not like to have a failure on his record and, therefore, perseveres. I do not want to suggest that reflective children's behavior is superior or better in all situations; it depends on the task. I believe that we are viewing in these data an aspect of a variable that has always been present in man's description of man. Hippocrates talked of the sanguine, choleric, and phlegmatic types, and Jung talked about introversion and extroversion. These terms capture a stable characteristic of human beings, and we think that the reflection–impulsivity dimension is related to these typologies.

DR. YOUNG: Did you find any intelligence differences between the reflective and the impulsive types?

DR. KAGAN: Girls show a higher correlation between reflectivity and verbal IQ than boys, and that is a consistent finding. But the correlation is not high enough to suggest that these are similar processes. Dr. Keogh has some interesting experiments that would help us to understand these data and that also relate to Dr. Sperry's ideas on cerebral dominance.

DR. KEOGH: Most of the traditional tests of spatial orientation have been paper-and-pencil tests. Youngsters are asked to copy a pattern to see whether they can produce something like the original. The child is usually sitting in a relatively stationary position, and the reference points are clearly defined for him. We have been asking children to walk, a three-dimensional activity, to reproduce some rather simple patterns, and it is very interesting to observe their behavior. There was a definite difference between boys and girls in the production of simple and complex forms. We asked our children to pretend they had paint on the bottoms of their feet, and to make a five-pointed star, or to make one in the sand by walking in it, which was something relatively easy for them to do. We noticed in an early study (Keogh and Keogh, Amer. J. Mental Def. 71:1009–1013, 1967) that normal British schoolboys aged 6–9 years showed improvement with age in their ability to make these patterns by walking. Later, in this country, where we were working with 8- and 9-year-old normal youngsters, we asked them to walk patterns under three conditions. One was to walk on the open floor with no reference points defined; they were told in essence, "You can walk anywhere in the room that you want to; make the pattern any size you want, in any position you want." Another was to walk the same pattern on a 9- by 9-ft plain linoleum mat; this condition limited the space to be used and supplied structure to the field. And in the third test, the mat was covered with very fine sand so that the footprints left a good pattern.

We found with boys that they got better under the three conditions and were best on the sand—they made very precise figures, with accurate angles and so forth. Some 8- or 9-year-old normal girls walked these patterns under the same conditions as for the boys. There were some differences in pattern orientation, and the boys were significantly better at producing precise figures (Keogh, submitted for publication).

There are many reasons why this might be so, but it is probably simply that girls just do not use the available cues. The differences were dramatic, not only in the precision of the patterns, but also in the sort of organizational system used. To be specific, one of the patterns was a triangle inside a circle. Almost without exception, the boys walked a circle, then stopped, sometimes stepping off the mat or the sand, and then made the triangle. We have observed that girls attempted to make the patterns in one continuous line as if it were all one big pattern; they did not seem to break it down into parts.

One of our patterns was two circles in a horizontal plane. We have had a number of girls, but fewer boys, who walked a figure "8." We feel we have come across something that is fairly specific in perceptual abilities, namely, a sex difference in spatial perception. All these children can discriminate between the patterns, and all can draw them.

We have also found (Keogh, Amer. J. Mental Def., in press) that retarded youngsters are very disoriented. One sometimes wonders how they ever get home, because they really do not seem to know where they are.

ROBERT L. FANTZ

Visual Perception and Experience in Infancy: Issues and Approaches

The intake and processing of information by young infants is a relatively new field of study, even though there has been a rapid acceleration of research efforts during the last decade. It is too soon to give a clear picture of the visual world of the infant or to attempt to state the basic principles underlying early perceptual development and acquisition of visual information. Certainly, it is too soon to select with confidence the findings that will prove to be of most value in understanding the eventual attainment, or lack of attainment, of complex perceptual performances by the child and adult. It will have to suffice to point out some of the issues in the field, to describe some promising approaches to them, and to present a highly selected sampling of data already obtained. The material to be covered is heavily influenced by the viewpoints of one investigator in the field and by the research that has recently been done in one laboratory—the Perceptual Development Laboratory of Case Western Reserve University.

The field of early perceptual development can be conveniently divided into four interrelated topics: visual–motor coordination and spatial localization, visual resolution and discrimination capacities, visual preferences and other selective responses to visual stimulation, and the retention of visual information and other effects of visual experience.

VISUAL-MOTOR COORDINATION

Because this topic is covered by other papers of this conference, my remarks here will be limited to emphasizing the distinction between the first two topics. Visual–motor coordination is the primary determinant of spatial localization and accurate directed responses, whereas visual discrimination capacity is the primary determinant of the intake of visual information, assisted by visual–motor coordination to the degree that it may exist. This distinction raises an issue of historical importance: By what response indicators shall visual perception be measured in the young infant? For many of the early investigators (e.g., Gesell *et al.*,[15] Ling,[23] and McGraw[25]) visual perception was operationally defined as the ability to successfully fixate, pursue, and grasp an object. Because these abilities were found to be initially poor or absent and only gradually perfected during the early weeks or months of life, the assumption was that other important aspects of visual perception, especially the ability to distinguish the form and patterning of objects, were similarly slow in developing. But lack of precision in oculomotor coordination or accommodation, or even severe impairment of these functions, does not preclude the ability to see, although it interferes with the most effective use of vision. This may be illustrated by the evidence from patients with congenital cataracts, who, soon after operation, sometimes were able to distinguish between (as distinct from naming) different colors and forms, in spite of lacking a lens, having minimal oculomotor practice, and often having spontaneous nystagmus.[28,30] Conversely, precise coordination in fixating, pursuing, and grasping an object does not indicate whether the form, size, color, solidity, or any other attribute of the object is discriminable. Good visual–motor coordination and spatial localization are as phylogenetically old as the vertebrate eye, but the ability to discriminate complex forms has only gradually evolved.

An obvious answer to the question of how to measure perception in the infant is that responses and experimental procedures appropriate to the particular aspect of perception under study should be selected. For measuring visual–motor coordination and spatial localization, the precision of the infant's response to a single stimulus target is sufficient. For measuring discrimination or identification in nonverbal subjects, a minimum of two targets is required, along with the usual controls necessary for a reliable discrimination experiment. The nature or precision of the response is not critical, as long as the subject's choice between stimuli differing in some dimension can be determined without ambiguity. The visual fixation response, in particular, is sufficiently coordinated even in the newborn infant to indicate the choice of one or another target. Recent studies,[2,5,18,19] with more accurate measurement and better-controlled stimulus conditions than most of the earlier work, have generally found the neonate to possess better-developed abilities to fixate, converge, pursue, and accommodate than had been realized.

VISUAL DISCRIMINATION

Visual-discrimination tests in nonverbal subjects have traditionally used discrimination training or other conditioning procedures. Until recently, these procedures were not thought to be applicable to the infant in the early months, owing to the lack of coordinated, visually directed responses, as well as to the lack of adequate learning abilities. But advances in technology have made feasible the conditioning even of newborn infants (see the following presentation, by Lipsitt) and have opened up wide possibilities for discrimination testing in young infants. In particular, Bower[1] was able to demonstrate in the 2-month-old infant a number of phenomena of spatial perception, including depth discrimination, orientation discrimination, size constancy, and shape constancy. He first conditioned, using "peek-a-boo" as reinforcement, a head-turning response to a particular object, and then tested for discrimination among this and other objects differing in specified ways.

Most of the available information on discrimination capacities, especially pattern discrimination, in the early months of life has come from the visual-preference method. This approach to discrimination testing measures the untrained differential responsiveness to two or more stimulus targets. The consistent tendency of the infant to look longer or more often at a particular target in repeated exposures, with position and other extraneous factors controlled, indicates both the ability to distinguish a stimulus difference and the selection of a particular stimulus for attention. Of course, the absence of a differential response may indicate either inability to discriminate or equal interest among the targets. This limitation has proved not to be serious, in view of the wide range of stimulus attributes that *have* elicited visual preferences.

The question asked in the early visual-preference experiments was how soon the infant began to see patterned stimulation, the source of most visual information. The controversy between those who argued that the infant requires a long period of visual experience and learning and those who insisted that the infant requires a long period of postnatal maturation was eventually resolved by the finding that even the newborn infant can resolve and discriminate patterns.[6,10,19,20,27] But there remained three issues: When does the visual system first become capable of function? What are the discrimination capacities at various maturational levels after that beginning point? What are the specific contributions of postnatal experience to the initial primitive level of perception? Research on the last of these will be discussed below. Regarding the first and second issues, some very recent work[26] has provided the most relevant information, as well as incidentally giving the most extensive and most reliable data available on differential visual responses in newborn infants. These results will be summarized here.

Miranda[26] tested a group of 27 premature infants, less than 38 weeks after conception but after various periods in an incubator, and compared them with 27 full-term infants averaging 3½ days of age. Our paired-comparison visual-preference technique[13] was modified to the requirements of the small and immature subjects. The infant was secured in a semireclining position in a form-fitting, adjustable seat and rolled under a stimulus chamber (Figure 1). The chamber was relatively homogeneous inside, except for two stimulus targets (mostly 5-in. squares) placed side by side 1 ft from the infant and about 1 ft apart from center to center. Each of 14 pairs of flat, achromatic targets (Table 1) was exposed for two 10-sec periods with reversed left and right positions on the second period. Vertical striped patterns of three widths were each paired with gray to determine the smallest pattern that would be discriminated from an unpatterned target, as a rough estimate of visual acuity.[14] Listed next are three steps in a "complexity" dimension, using a series of white squares containing 0, 1, 4, or 16 black squares, regularly arranged. In the next three pairs, linear versus curvilinear forms or arrangement were opposed, but with white : black ratio and length of contour equated. The remaining five pairs of assorted and often multiple stimulus variations were taken from previous studies indicating differential responses by newborns or infants in the early weeks of life.

The reliability of the experimenter, who observed through a tiny hole

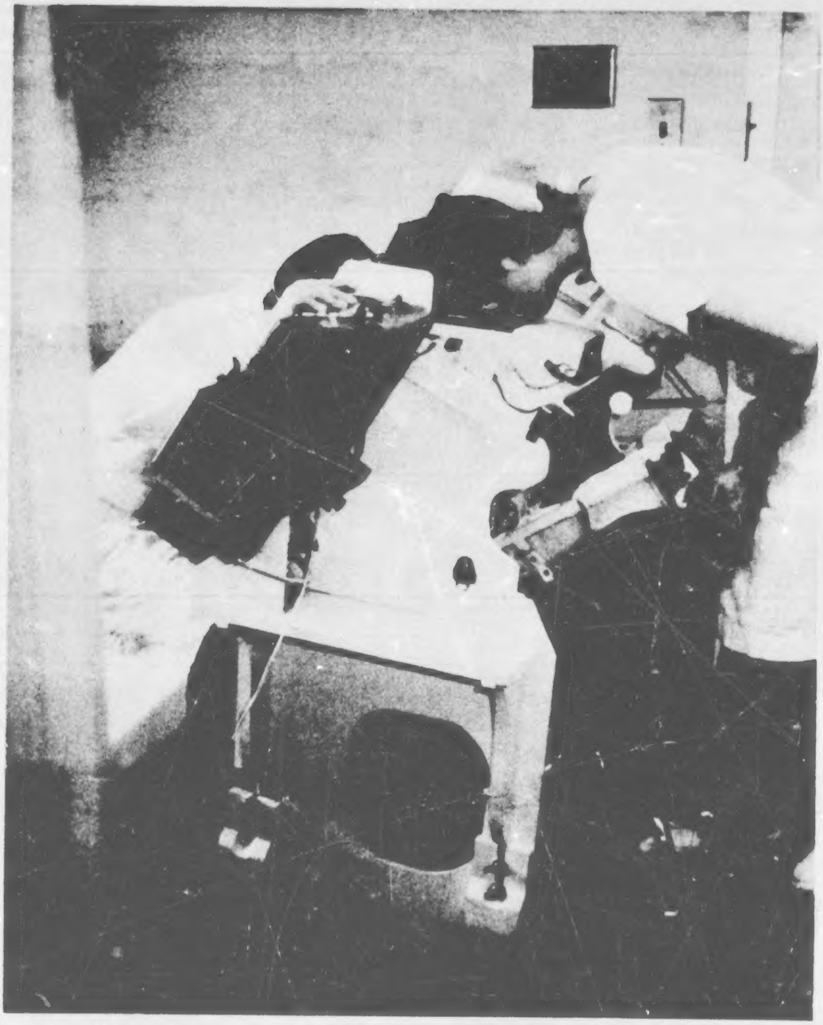

FIGURE 1 Visual-preference testing apparatus in mock operation. So that the baby could be seen in the picture, the crib was not set in as far as it would be in normal operation. The experimenter observes through a peephole between the targets. The second observer (standing) looks at a mirror above this hole. The recording unit and timer can be seen in the foreground. (From Miranda.[26])

TABLE 1 Differential Visual Responses of Premature and Full-Term Newborn Infants[a]

Stimulus Targets	No. Infants Showing Longer Fixation of Left or Right Stimulus of Pair[b]	
	Premature[c]	Full-term[d]
Acuity gratings		
$\frac{1}{2}$-in.-striped-gray	*22-0*	*26-0*
$\frac{1}{4}$-in.-striped-gray	*23-1*	*23-3*
$\frac{1}{8}$-in.-striped-gray	13-7	10-11
Complexity pairings, no. of elements		
0-1	*2-21*	*3-24*
1-4	*18-9*	15-11
4-16	20-7	16-8
Linear versus round configurations		
L1-R1	14-7	13-11
L2-R2	12-10	12-11
L3-R3	9-10	13-10
Other stimulus variations[e]		
1A-1B	*22-5*	*19-6*
2A-2B	15-5	17-9
3A-3B	*21-3*	*20-2*
4A-4B	*24-1*	*25-0*
5A-5B	7-11	13-10

[a] Adapted with permission from Miranda.[26]
[b] Tie scores and incomplete tests are omitted; results in italics are significant ($p \geqslant 0.05$) by two-tailed correlated t-test on time scores transformed for parametric analysis.
[c] Mean conceptional age, 35$\frac{1}{2}$ weeks; mean postnatal age, 22$\frac{1}{2}$ days; gestation varied from 28 to 37 weeks.
[d] Gestation was over 37 weeks, with a mean of 40 weeks; age, not more than 1 week.
[e] Top to bottom pairs shown in Figure 3 as pairs 1, 13 (with circles replacing the oval outlines), 17, 8, and 5.

between the targets, in recording fixations was checked for most of the subjects by a second observer. This observer looked through a hole in the ceiling of the chamber into a small mirror above the center hole, adjusted to reflect the eyes of the infant, and recorded without knowledge of the targets or of the experimenter's recordings. In spite of inferior observation conditions for the second observer, the results for the two were in good agreement, in that the same pairs of targets were found either to show or not to show differential fixation, and the median of the discrepancies between observers, disregarding the sign of the difference, was less than 2 sec in 456 tests of 20-sec duration.

Table 1 gives one measure of the differential responses: the frequency of longer fixation on one or the other target of the pair. Significant preferences were shown by both premature and full-term groups for six of the pairs: the two larger acuity patterns over gray, one square over plain white in the complexity series, one of a pair of diverse black-and-white patterns (1A versus 1B; pair 1 in Figure 3), a large over a similar small pattern (3A versus 3B; pair 17 in Figure 3), and a sharply defined pattern over one with shades of gray (4A versus 4B; pair 8 in Figure 3). Significant preference was shown by the premature infants alone for an additional pair (one over four squares).

It is evident from the responses of the premature sample that the various parts of the visual system are capable of function at least a month before the usual time of birth. This conclusion is not altered by the possibility that the longer opportunity for postnatal experience and oculomotor practice for the prematures had some facilitating effect on the visual performances. However, such an effect, tending to compensate for the shorter period of maturation, is one of the possible explanations for the surprising degree of similarity in the responses of infants that have had 8 or 9 months to develop. It might even appear that the premature infants showed *better* pattern discrimination for several pairs. But at least in the case of the complexity pairings this is illusory, for the lower differential shown by the full-term infants probably represents the beginning of an upward shift in the preferred or "optimal" complexity level. Evidence of such a shift was obtained by a significant negative correlation between the degree of preference for the one-square over the four-square pattern and the maturational level (age from conception) of the combined premature and full-term groups. This result hints of the issue, to be discussed later, of whether the age of the infant should be measured from conception or from birth in developmental research.

On the stimulus side, it is evident from these results that both premature infants (of the given range of conceptional and postnatal ages) and full-term neonatal infants can discriminate between patterns and plain surfaces of the same size and light reflectance, and that they can discriminate between different patterned surfaces in some cases. The results cannot be considered to indicate the limits of pattern vision in either group, unless the optimal testing conditions and stimulus variations are known. For example, better visual acuity than hitherto found has been suggested for newborn infants by tests using both differential fixation and optokinetic nystagmus responses, but with different ranges of pattern size and other differences in conditions.[5,10]

These and other results from newborn infants give no conclusive evidence of ability to discriminate between two patterns that are different in form or arrangement but equated in "complexity" or "quantity," such as the linear versus round pairs of Table 1. But for slightly older infants, such an ability has been demonstrated repeatedly: differentiation between irregular and regular arrangements of squares was suggested within 2 weeks of age,[10] between stripes and bull's-eye patterns during the first month,[7] between linear and circular configurations of line segments by 5 weeks, and between lattice and checkerboard arrangements of squares by 7 weeks[13] (see Figure 3, pairs 3 and 5). Another study[12] used a set of four different arrangements of 25 white squares against a dark background, with three of the arrangements being derived from a five-by-five matrix and differing only in orientation of the elements. Differences in response time increased with age (Figure 2) and became significant starting with the group at the age of 1–2 months.

It may be concluded that within the first 2 months of life the infant can discriminate among patterns equated for total area, light: dark ratio, number of elements, and length of contour. On the other hand, it may be concluded that infants can discriminate from birth among patterns differing in such measures as number of squares in the pattern, length of contour, and number of "turns" in a random shape.[3,19,20,22,24] Older infants have often but not always shown higher preferred-complexity levels. Without getting into the problem of defining "complexity" and distinguishing it from other variations in patterning, it can safely be stated that the young infant can discriminate more than one dimension of visual patterns.

These various developmental changes in responsiveness to patterns might be explained by improvement in visual acuity, by an increase in the number and variety of discriminable dimensions of patterns, or by changes in the preferred points along the discriminated dimensions. The resolution of finer patterns with increasing age very likely contributes to some changes, but it would not explain demonstrated changes in preference between patterns containing the same width of lines or size of squares; furthermore, the patterns in most studies have all been well above threshold. Regarding the second possible explanation, additional stimulus characteristics undoubtedly become discriminable with age

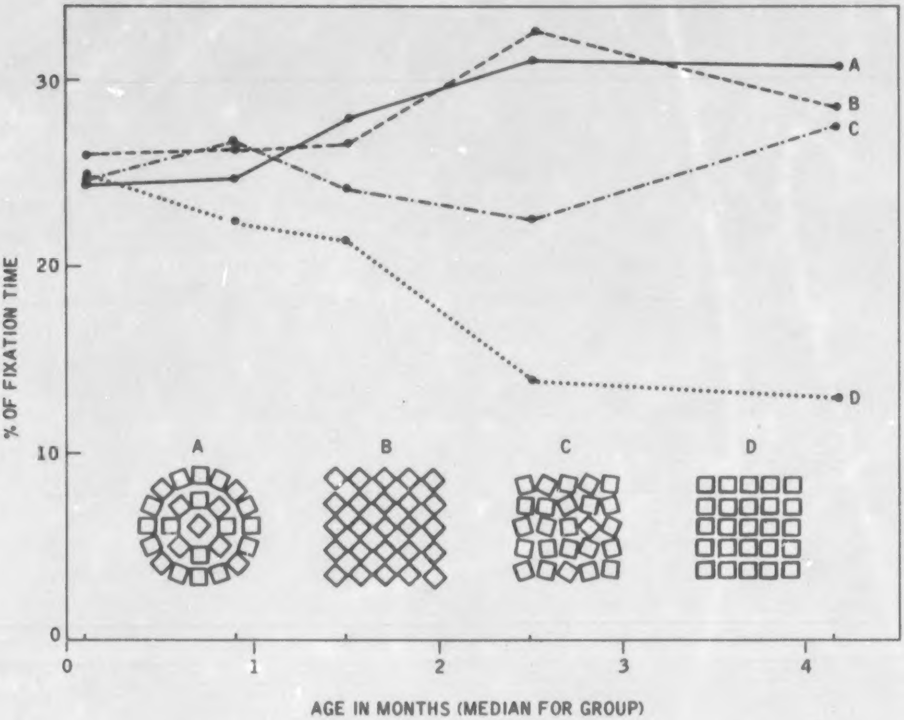

FIGURE 2 Relative visual attention to four arrangements of white squares, averaged for infants in five age groups. (Reprinted with permission from Fantz and Nevis.[12])

and experience (see Gibson[16]) and may result in some preference changes. But this explanation is not plausible when the patterns are found to be equally discriminable before and after the change, as in the case of a shift in preference from one to another of two patterns differing in complexity or configuration. Thus, some of the changes cannot be attributed to the development of visual capacities as such, and indicate, instead, the increased attention value of some patterns relative to others. Changes in visual selectivity may have as much portent for perceptual development in the infant and child as changes in visual acuity.

VISUAL SELECTIVITY

Optimal perception of an object requires the turning of the eyes so as to allow foveal examination of the object. This involves picking a small area from a large, intricate, and variegated visual array. At a given moment, this selection might be due to the intrinsic attraction of some stimulus characteristics; it might be a temporary interest in a stimulus due to novelty or movement or proximity; or it might be due to a long-term familiarization or conditioning process. Visual–motor and discrimination capacities set the limits of what the infant *can* perceive, but the nature of the environment and the selectivity of the moment determine what *is* perceived and hence what is learned about or responded to. The infant's experience is influenced just as surely by a tendency to look four times as long at one stimulus as at another as it is by the presence of one stimulus as opposed to another. Therefore, it is important to know the basis of selection by the infant and how it changes with age. For this purpose, the visual-preference method offers a simple and direct approach, although other relevant response indicators, especially for autonomic responses, can measure the "intensity," rather than the duration, of attention.

The most general finding for infants of all ages is that a plain, nonmoving, unpatterned surface or object of any color or brightness is low in attention value; it is probably more than coincidental that such a stimulus conveys little information. Conclusions as to the characteristics of nonmoving stimuli that are of high attention value depend much more on the age of the subjects and on the particular stimuli available. Among the stimuli that have been used to date, infants during the early weeks of life have looked most at sharply defined patterns, especially black-and-white patterns. That was brought out most clearly in a longitudinal study designed for other purposes—namely, to show differences in the rate of development of visual preferences between two selected groups of infants.[12,13] The 18 pairs of stimulus targets (Figure 3) were chosen as those most likely, on the basis of past results, to elicit definite intrapair changes in preference within the first 6 months of life; the specification of relevant stimulus characteristics was not the aim. Results for some pairs will be given later. At the moment, the comparison of the responses among the 18 pairs is of interest to bring out the relative attention value of broad categories of stimulus targets. It happened that eight of the stimulus pairs included black-and-

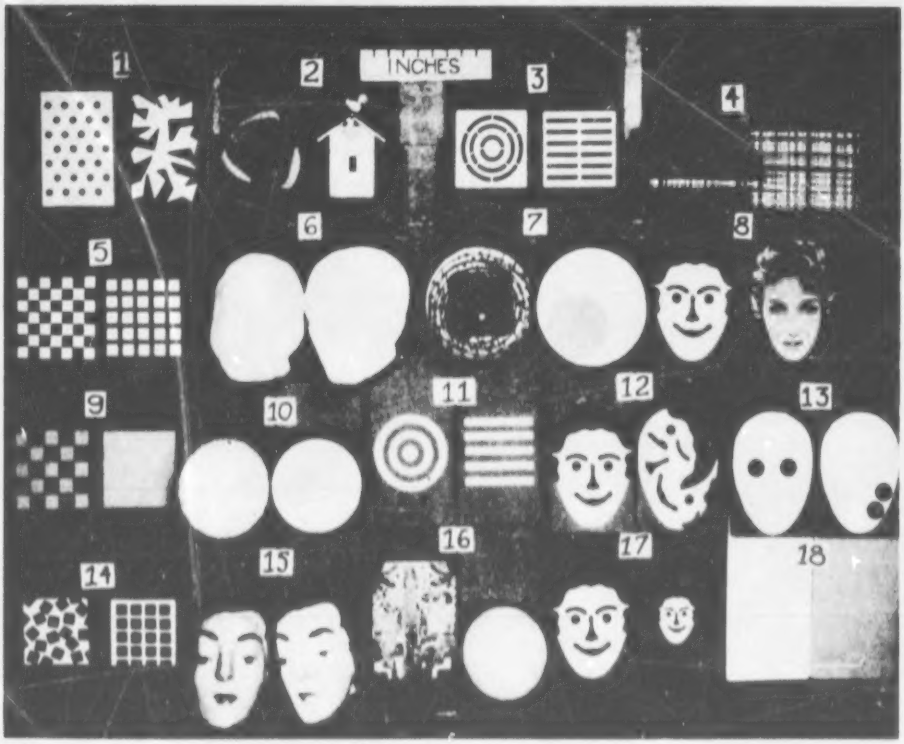

FIGURE 3 Pairs of stimulus targets used in the longitudinal study of selected home- and institution-reared infants, numbered according to order of presentation during each weekly test. Results in text are comparisons among the pairs and between the two members of some pairs. (Reprinted with permission from Fantz and Nevis.[12])

white patterns (this includes repeated testing of pair 1 following pair 12 and it includes pair 8, only one of whose members is black-and-white). Each of the eight pairs was fixated longer than any of the remaining pairs in averages of the seven tests given during the first 2 months of life. The ranking of these pairs, starting with the highest response, was pair 12, 1, 1 (repeated), 8, 17, 14, 3, and 13. The next two highest pairs in average fixation time (pairs 11 and 5) were also sharply defined patterns consisting of white elements against a blue background. By far the lowest in attention value was pair 18 (plain white and gray); the next lowest were pair 9 (bright red checks and square), then pair 16 (flickering orange light and complex pattern), and then pair 4 (red patterned boards). On the average, the eight black-and-white patterns were fixated significantly longer than the remaining pairs (even excluding pair 18) for each week of age from 2 through 12. However, this differential decreased considerably after 8 weeks; it disappeared by 15 weeks, and was reversed later. The pairs containing facelike patterns were initially about as high in attention value, and dropped almost as much, as the abstract patterns.

It would seem that both the complexity and the configuration of a pattern are less critical determinants of visual attention early in life than its contour definition or brightness contrast. Although the simplest black-and-white pair of patterns (pair 13) was the lowest ranking of the eight, it was fixated longer than many pairs of targets that would be rated more complex by any definition. After 2 months of age, high pattern definition was not sufficient, although some sharply defined patterns continued to receive long fixation times. Between 4 and 6 months, widely diverse targets were of high attention value; the top-ranking five were pair 7 (mesh and wood object versus rotating red-on-yellow disk), pair 8 (schematic face versus face photograph), pair 15 (solid head model versus photograph), pair 16 (Egyptian art versus flickering orange globe), and pair 2 (brightly colored toys).

Visual selectivity and developmental changes were still more marked in the comparison of the lengths of response to two stimulus targets exposed together. The selected results presented here are based on reanalysis of published longitudinal data on the first 6 months of age from the 10 infants constituting the "home sample,"[12,13] combined with data from follow-up tests of the same infants during the second 6 months of life. In Figures 4–9, the graphed fixation-time averages before 16 weeks of age are pooled averages for two successive weekly tests; after that age, they represent single tests given every 2 weeks through 24 weeks of age and every 4 weeks thereafter. The target designations are based on left or right position of a stimulus as shown in Figure 3; testing positions of the two were varied systematically within each test and among successive tests.

The results for two pairs of sharply defined, abstract, achromatic patterns are given in Figure 4. There is a general decrease with age in response to these patterns, as indicated above. More striking, however, is the rapid development of preference both for the bull's-eye pattern

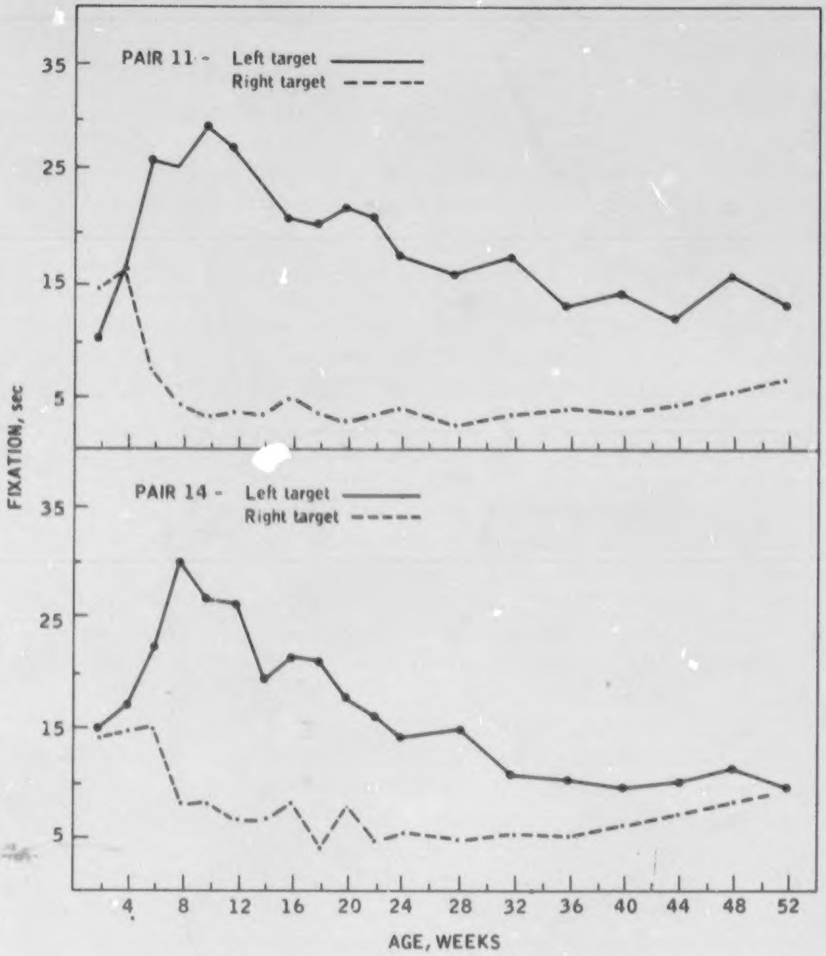

FIGURE 4 Fixation times for each target of a pair presented together for a total of 40 sec, averaged for the same 10 infants at each age. Left and right targets are as illustrated in Figure 3. Both pairs were flat, achromatic, sharply defined patterns varying in circular versus linear contours (pair 11) or in irregular versus regular arrangements of squares (pair 14).

and for the irregular arrangement of squares, reaching a peak in the third month, and then gradually falling off.

Differentials for other pairs were less strong and consistent, even when the stimulus difference was much greater. For example, in Figure 5, the targets preferred during the first month of age and then again after 5 months were (in pair 7) a slowly rotating disk covered with fluorescent red and yellow paper and (in pair 16) a flickering 40-watt orange light bulb enclosed in a 6-in. translucent globe. The targets preferred between 2 and 4 months were much higher in informational value but would generally be considered to be uninteresting: a spiral of wire mesh mounted to a board with holes in it and partly painted white (pair 7) and a dark achromatic photograph of intricate Egyptian art (pair 16).

In Figure 6 (top), the strong initial preference for a black-and-white schematic face pattern over a face photograph disappeared by 16 weeks of age, most likely owing to the decrease in the early attention value of sharply defined patterns and the subsequent interest in other, more subtle patterns—as was true for pair 16. The irrelevance of the facial resemblance is suggested by an absence, in the lower graph, of preference for the correct over the scrambled arrangement of a schematic face until about 20 weeks of age; in fact, there was some early preference for the scrambled face. These results are not in agreement with those in various studies of institution infants that indicate a general preference for the correct arrangement during the third month.[10] However, the differential for this pair has never been as strong or consistent as that repeatedly obtained with pairs of geometric patterns (*cf.* Figure 4). Other investigators' results have also varied as to whether or when infants discriminate correct from distorted face-like configurations, even though face-like stimuli in general have been of high attention value. It is probable that by several months of age the infant is capable of discriminating the various experimental representations of faces both from each other and from real faces and that the presence or absence of differential attention to some representations has little relevance to social recognition and responsiveness.

Figure 7 illustrates the visual selectivity found in a number of studies relative to two further types of stimulus variation. The top graph shows the development of increased responsiveness to a solid, contoured object, along with a corresponding decrease for a flat surface. Although the solid object, to an adult, resembles a sculptured head without

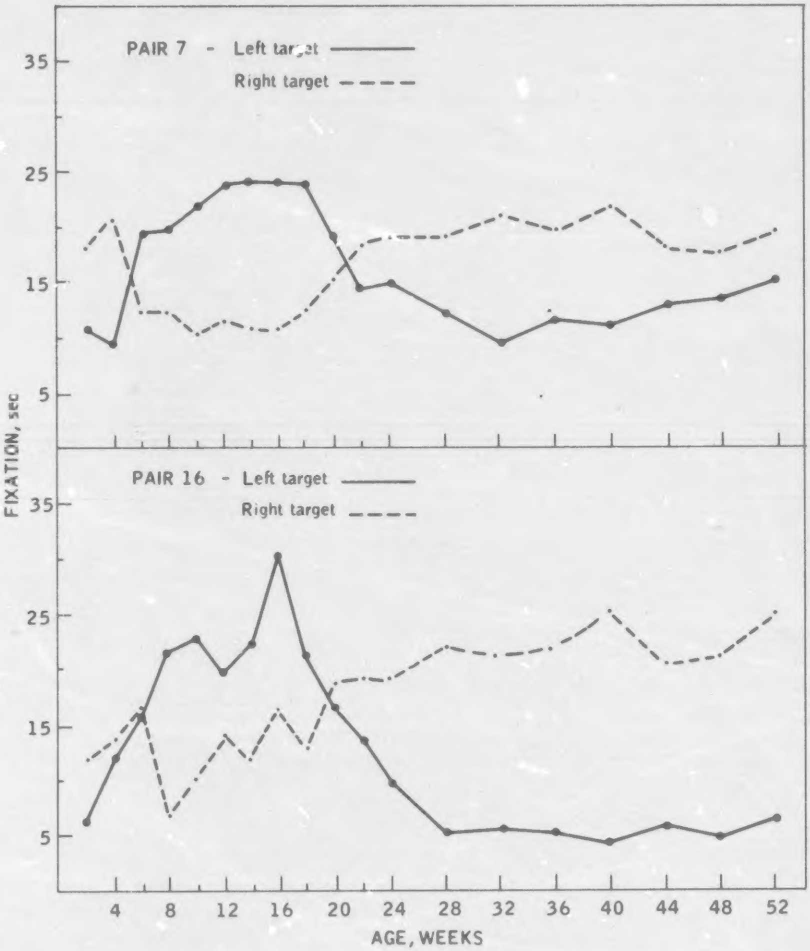

FIGURE 5 Fixation times for each target of a pair presented for a total of 40 sec, averaged for the same 10 infants at each age. Targets (from Figure 3) were a complex achromatic object (pair 7, left) versus a plain, brightly colored, rotating disk; and a dark achromatic art reproduction (pair 16, left) versus a blinking orange globe.

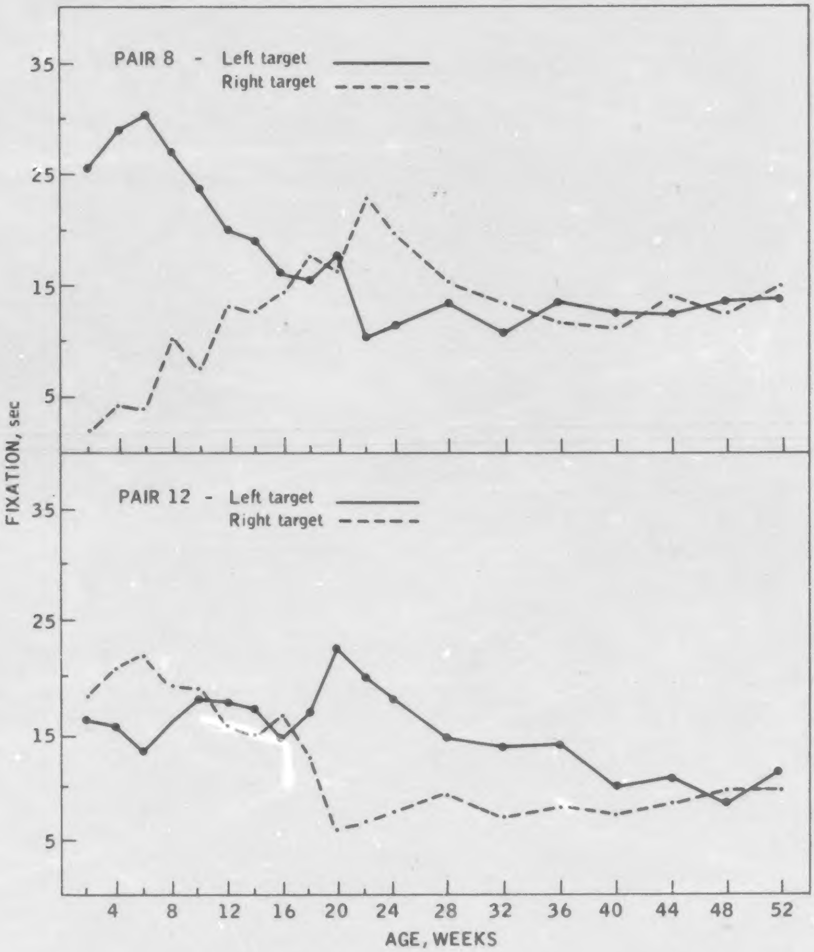

FIGURE 6 Fixation times for each target of a pair presented for a total of 40 sec, averaged for the same 10 infants at each age. Targets (from Figure 3) were schematic face pattern (pair 8, left) versus a face photograph; and the same schematic face pattern (pair 12, left) versus a scrambled arrangement of the same features.

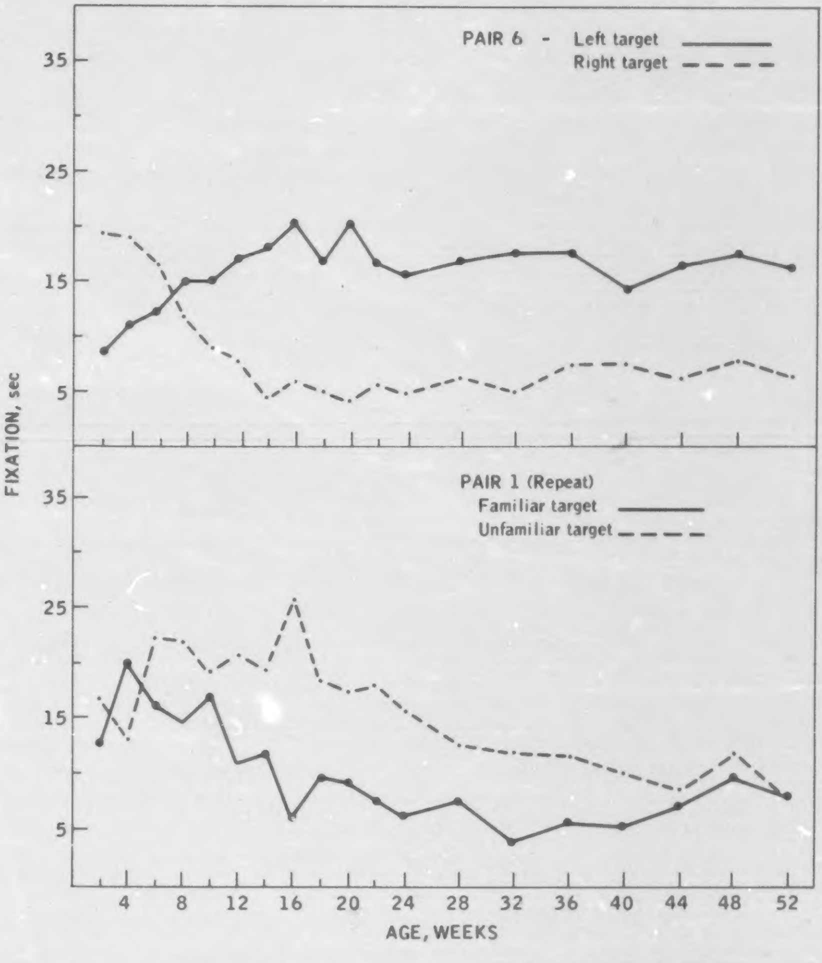

FIGURE 7 Fixation times for each target of a pair presented together for a total of 40 sec, averaged for the same 10 infants at each age. Targets (from Figure 3) were a solid model of a head, painted flat white (pair 6, left) versus a similar white outline form; and one of two patterns (varied from week to week) that had been repeatedly exposed to the infant during the course of a testing session (pair 1, familiar) versus the other pattern, which was relatively novel.

painted features, the preference development is closer to that shown for other variations in solidity than that for other face-like pairings. As with several other pairings of solid and flat targets, for pair 6 the solidity preference appeared in the early months and was maintained thereafter.

The bottom graph of Figure 7 shows the development of a preference based on specific visual experience, rather than on either intrinsic stimulus characteristics or general types of familiar objects (such as solid objects or face-like patterns). One of a pair of diverse black-and-white patterns was placed in such a position in the stimulus chamber that it would be exposed during the 10 sec or so after each test exposure—that is, while the targets were being reversed or changed. After 22 such exposures, this pattern was tested along with the other pattern of pair 1. The two patterns were used as the "familiar" patterns on alternate test weeks for each subject. Although the novelty preference was not strong, compared to many stimulus variations, it was consistent among the infants after 2 months of age and for most of the first year, before it finally disappeared—perhaps due to the decreased interest in flat patterns in general.

Results such as these, showing a change in responsiveness to repeatedly exposed patterns, are of direct relevance to the ability to process and retain visual information. Such results might well have been included under the heading of "visual experience." But they are equally relevant to visual selectivity, inasmuch as novelty is one category of stimulus determinant of differential attention—one that happens to be of particular theoretical importance. Surprisingly, response to novelty has been quite difficult to measure reliably and predictably; it has been affected by seemingly minor variations in experimental conditions.[8] One of the persistent problems is that of equating patterns in initial attention value, so that effects of recent exposures will not be obscured. Another problem is to ensure that the decrease in response is specific to the pattern and therefore indicates pattern recognition. Some studies have not controlled this factor, and their results may indicate only a general habituation of response, decreased arousal, or fatigue. One solution is to use a paired-comparison procedure and to determine the response to the familiar stimulus relative to a novel one of similar attention value.[11,13]

There are other solutions, however. For example, Caron and Caron[4] used the single-stimulus procedure quite effectively. They demonstrated the specificity of the response decrement for a repeated pattern by the maintained high response to novel patterns. The same study showed a smaller response decrement for a complex than for a simple repeated stimulus and showed retention of visual information over a short interval–i.e., a single presentation of the familiar stimulus following three novel stimuli brought a significant drop in fixation time. These and other characteristics relevant to the effects of specific short-term visual exposure have only begun to be explored.

Two additional variables, the source of the subjects and the duration of the test exposure, were found to be related to short-term familiarization effects. In the longitudinal study using the stimulus pairs of Figure 3,[12,13] two samples of infants were selected so as to be as different as possible in parental background and early environment. Half the infants were offspring of university faculty, the others were institution-reared infants. All infants were healthy, full-term, and of Caucasian parents; the mean birth weights of the two groups were identical. The familiarization test, included as part of the weekly preference testing, was as described above; fixation times for the home group were as given in Figure 7. To permit better comparison of the two groups, fixation times were converted to percentages of total fixation time given to the familiar pattern in successive testing weeks (Figure 8); 50% indicated chance response.

The top graph, based on the entire 40 sec of postfamiliarization exposure, shows decreasing response to the familiar pattern with increasing age for both groups. This development was more rapid for the home group; the difference in percentage between groups was significant at 12 through 18 weeks of age. In a subsequent analysis of these data by successive 4-sec periods of exposure, it was noticed that in the early months the home infants showed more differentiation between familiar and novel patterns after longer exposure to the two, as though recognition of the familiar pattern (or habituation to it) required some period of examination of both patterns. The institution infants did not in the early months show this change during the exposure. Consequently, the preference curves based on only the last 4 sec of the second 20-sec exposure (bottom graph) show more group difference and show a significant novelty preference by the home group as early as 6 weeks of age.

Increased stimulus differentiation after longer examination was not the usual outcome. For most of the pairs, varying in intrinsic stimulus characteristics rather than in previous exposure, at least as much prefer-

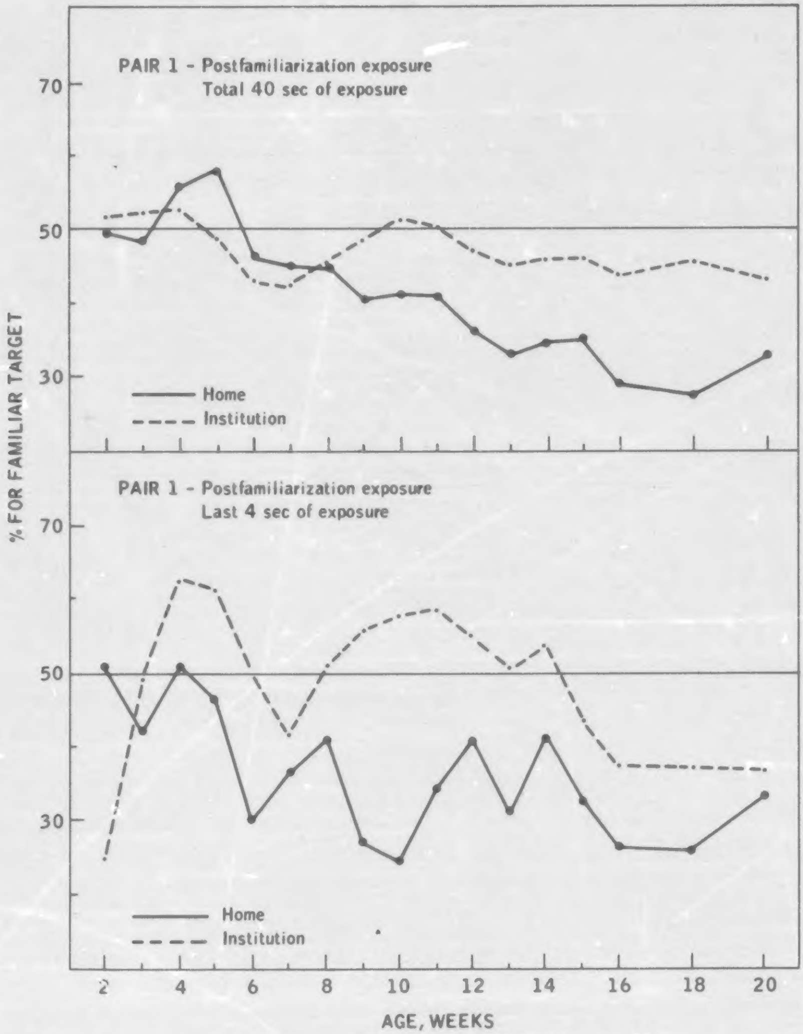

FIGURE 8 Pattern familiarization results during the first 6 months for the selected home-reared infants of earlier graphs compared with 10 institution-reared infants. For each group, fixation times for the two targets were converted to percentages for the target that had been repeatedly exposed earlier in the testing session, relative to the novel one. Percentages are based either on two 20-sec exposures (top graph) or on only the last 4 sec of the last exposure (bottom graph).

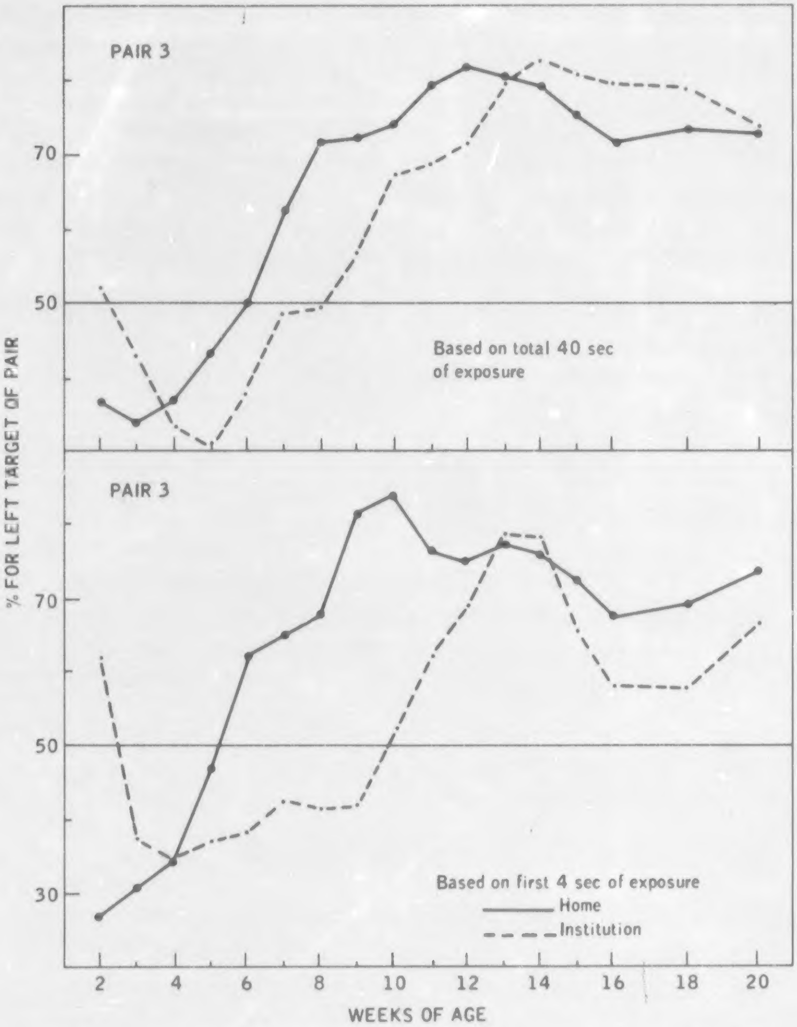

FIGURE 9 Results comparing selected home infants and institution infants on development of preference for circular over linear arrangement of line segments. Percentages of fixation time are for target pictured on left (Figure 3), based either on two 20-sec exposures (top graph) or on only first 4 sec of the first exposure (bottom graph).

ence was shown early in the exposure of a pair as later on, especially by the home group. Consequently, in a few cases, more difference between groups was shown in preference curves based only on the first 4 sec of exposure, as will be illustrated with results for pair 3.

The development of preference for a circular over a linear configuration of line segments (Figure 9) is one of the best examples of the difference between the groups in the age of preference development. For the total 40 sec of exposure (top graph), the two curves are almost identical in shape but are displaced about 2 weeks in age, indicating earlier development of a basic perceptual response by the selected home-reared group. For the first 4 sec of the first 20-sec exposure (bottom graph), the group difference was still greater: the home infants appeared to be able to discriminate the patterns within a few seconds as well as after long examination, whereas the institution group appeared to require more time for examination to show maximal differential response. The accentuated group difference for the first 4 sec of exposure is only suggestive; it was not duplicated for any other pair of targets. These and other less clear-cut results indicate that tracing changes in visual selectivity within short exposures, as well as over weeks of age, is a promising approach to the problems of studying information processing and effects of visual exposure in the young infant.

In the overall analysis of group differences, including the results given in Figures 8 and 9, the home group of infants showed significantly earlier or greater changes in preference for eight pairs of targets taken separately and for all 18 pairs averaged together.[13] The significance of changes in visual selectivity as indicators of rate of perceptual–cognitive development is suggested by this differentiation between groups of infants with high expectation of eventual differences in cognitive performances, due to either congenital or early environmental differences. This significance is also suggested by the scores on the Griffiths Mental Development Scale (modified for better appropriateness for institution infants) given at 20 weeks of age. These scores for the combined groups showed a correlation of 0.74 with the rate of preference development during earlier months of life. In contrast, the scores during the early months, measuring largely motor development and sensorimotor coordination, were not related to the preference changes shown during that period; in particular, the institution infants developed oculomotor coordination as early as the home infants. If early cognitive abilities are to be related to later intelligence, then "perceptual intelligence" appears to be a more appropriate description than "sensorimotor intelligence" for these early abilities.

VISUAL EXPERIENCE

A general finding of the visual-preference research has been that many of the basic skills and selectivities required for pattern perception are present at birth or within several months of age. Rather than arguing for a purely nativistic or maturational view of perceptual development, this finding instead emphasizes the role of experience by showing that visual exploration and information processing begin at birth and that ample opportunities for a wide variety of experiential effects are therefore present long before the infant is able to learn about the environment through manual or locomotor explorations. The main issues, then, are to what degree and in what way these opportunities are used.

As in the case of studying visual capacities, the available approaches to studying experiential effects are limited by the slow motor development of the infant, with the additional problem of varying experience in acceptable but sufficiently controlled ways. Recently developed conditioning procedures (as described above) can be of value, provided that the experimenter goes beyond the initial shaping of a response and is able to determine the specific stimulus features that have been perceived and remembered. For example, in the experiments of Bower,[1] the initial conditioning of head-turning to a particular object showed only retention of the response-reinforcement contingencies and discrimination between the presence and absence of an object. But the later discrimination tests with varied objects or situations showed that such features of the object as size, distance, shape, and orientation were perceived and remembered for a short time.

The experiments described above on decreased attention to a specific, repeatedly exposed pattern provide another approach for studying short-term memory, differing mainly in that the response tendency was initially present without conditioning and that it *decreased* with experience. This experience effect assumes added interest from the fact that the necessary conditions are no doubt frequently present in the everyday life of the infant and from the adaptive consequences that the infant spends more time looking at the relatively novel parts of the environment.

Long-term effects of experience are more difficult than short-term effects to study experimentally in human subjects. Changes in visual preferences over some weeks or months of age can indicate long-term effects if there is sufficient evidence for assigning an experiential cause. This was not possible for most of the changes described here, although experience may often have been important. In the area of depth perception, however, several lines of evidence converge to show the importance of experience in the development of the preference for solid over flat objects found to develop around 2 months of age (Figure 7).[10,13]

In research with infant monkeys,[9] it was found that subjects reared in darkness from birth until various ages required a period of living in a lighted environment for the development of preferences for solid over flat objects (comparable with those shown by human infants), but failed to develop consistent preferences if the deprivation period was over 6 weeks. In contrast, preferences for patterned over plain targets were shown initially without visual experience and then were lost with continued deprivation.

For varying experience in human infants, enrichment procedures must be substituted for visual deprivation, perhaps starting with infants who happen to be in a relatively stimulus-poor environment.[29] In a recent unpublished study of institution-reared infants using this approach, we provided 10 weeks of extra patterned visual stimulation in the crib and nursery (including a merry-go-round of varied objects) for every other entering neonate. In the results from weekly preference tests, for each of three pairs of solid versus flat targets the enriched group showed more attention than the control group to the solid object at one or more testing weeks. Few other significant differences in preferences were shown by the two groups, thus suggesting a limited area of effect of the perceptual enrichment.

In the same study, data were obtained on another variation in visual experience–one occurring naturally as a result of various lengths of gestation (within the normal range). For infants of the same conceptional age (i.e., length of gestation plus age from birth), an infant born several weeks before term, for example, has had that much more opportunity to see the world and practice visual coordinations than the full-term infant; the question is whether at that developmental level the extra experience is effective in some measurable way. The finding was that, for two pairs of solid versus flat targets, the development of a solidity preference was in closer agreement among the infants when their ages from birth were used than when their ages from conception were used, suggesting a determining role of early experience, regardless of the maturational level. However, many of the changes in pattern preferences were in closer agreement among the groups when ages were figured from the estimated date of conception (based on mother's report of last menstruation), suggesting the importance of degree of neural maturation for those responses.

It seems clear that both the quantity and the quality of visual experience determine to a considerable degree the beginnings of depth perception in the early months of life. Methodologically, the various results given above have demonstrated several practical approaches through which specific long-term or short-term effects of visual experience can be investigated, starting from birth. The simple procedure of comparing visual preferences or other discriminative responses at equal conceptional ages, as well as at equal postnatal ages, is particularly promising for distinguishing between experiential and maturational influences in the early months of life. One might argue that the usual practice in behavioral studies of considering only the age from birth implicitly assumes that nothing happens in the last weeks of fetal development to affect neurologic or behavioral maturity. By several years of age, the proportionally small differences in length of gestation within the normal range might well be ignored; but at birth or at several months of age, this practice sometimes may leave the pivotal variable out of the developmental equation and on other occasions may, through assuming complete experiential determination, throw away the proof of some effects of experience.

STAGES OF PERCEPTUAL DEVELOPMENT

In most of the research cited here, visual preferences have served as convenient behavioral indicators of the early development of visual perception and information processing. From another viewpoint, the visual selectivities themselves are part of the development process; they have considerable influence on what information is taken in for processing and for directing behavior. It is to be assumed, then, that in "normal" development the various changes in visual selectivities will tend to facilitate, rather than retard, the development process. And yet it is difficult even to guess at the adaptive value of all the changes in selectivity at various ages.

It would make good sense if the infant, starting with the selection of stimuli of minimal informational content, would gradually select more complex, more detailed, more subtle, more varied, more novel, and more changing stimuli as his capacities developed and his optimal complexity level rose. It might also be expected that this process would culminate in the fine discrimination and absorbed attention to patterned stimulation necessary for reading and other difficult perceptual tasks. The findings in the early months in some respects agree with this hypothetical view: the infant, although starting well above the minimal complexity level, does begin to attend to more complex, subtle, detailed, and novel patterns. But in later months, the attention value of various patterns generally decreases, and attention turns more to solid objects, flashing lights, brightly colored objects, and moving targets (among the targets used to date). By the end of the first year, little interest is shown in flat patterns and little differential attention is shown for differences among such patterns–a development seemingly opposite to the expected direction.

Although the accumulation of information by the developing child is a gradual and continuous process, the kinds of information received and the ways in which they are collected can change radically with age and experience. The young infant's absorption in patterns and differentiation of variations in patterning, reaching a peak in the third and fourth months of life, perhaps represents a stage in perceptual development in which the motto is "information for its own sake." In this stage, the infant spends vast amounts of time in seemingly useless visual explorations and examinations, guided by the selective tendencies of his maturational and experiential level. What is learned in this way is largely a mystery, but it is certain that it goes beyond the perfection of oculomotor skills and discriminative capacities and, most essentially, involves the accumulation or assimilation of information from visual patterning. Also, it is safe to assume that this stage is essential for normal progress toward stages of visual information processing. Interference with this stage is a likely factor in the retarding effects of environmental deprivation in infants and of complete deprivation of patterned stimulation in young animals.

One closely following stage is that in which visual exploration is a means, rather than an end; looking is a guide to oral, manual, and locomotor explorations and manipulations of the environment through which very different kinds of information are accumulated or assimilated. For this purpose, the patterning of flat surfaces is of little value (unless accompanied by tactile qualities), whereas object solidity and form are highly relevant stimulus characteristics that were found to continue to be of high attention value throughout the first 6 months of life. This apparent development has also been shown to take place in infant monkeys,[9] which in the early months of life without deprivation showed visual preferences for patterned over plain stimuli and for some patterns over others. Early in the second year of life, when given a series of discrimination-training problems preceded by "free-choice" tests, the animals showed few selective responses to, and subsequently rarely learned to discriminate, painted variations in color, brightness, or patterning. However, targets differing tactually as well as visibly (i.e., differing in form, texture, solidity, or size) tended to be differentiated in the free-choice tests; in discrimination training, these pairs all received more correct responses and were usually discriminated to criterion. The monkeys had learned in everyday life to pay attention to palpable, "real" object characteristics and to ignore differences between painted surfaces that were irrelevant for behavior. The variations in patterning or color of flat surfaces were still discriminable in the sense of being within the known capacities of the animals; that had been shown by early visual preferences. But they were not actually discriminated at this stage because they were not noticed.

It may be asked whether the early selectivity for patterns and the differentiation among patterns has any adaptive significance, inasmuch as it is replaced by other selectivities some months later. Perhaps the early selectivity for patterns is simply the consequence of the selectivity of the nervous system: the high responsiveness of visual receptors and nerve cells, especially cortical cells, to spatial or temporal patterns and the low responsiveness to uniform stimulation may in the early months be directly translated into oculomotor responsiveness. This explanation is certainly plausible in general, and it could be related to some of the specific pattern selectivities shown by the infant. For example, the tendency to give more attention to regular, linear patterns than to various other configurations during the early weeks of life could be a direct behavioral expression of the selective responsiveness of cells in the visual cortex to linear contours.[21] Also, subsequent shifts in preference to circular or irregular configurations could signify the dominance of higher levels of neural organization in the direction of attention, but this is only a vague conjecture, made in the absence of other explanations of the amazingly strong selectivity for some pattern configurations in terms of either adaptive value or experiential origins.

However, the more general selectivity for patterns in the young infant can easily be considered to be adaptive in focusing attention on information-rich parts of the surroundings and thus facilitating the process of perceptual learning. Developmental shifts in preference to more complex patterns, to more subtle variations in patterning, and to novel patterns appears to be similarly adaptive. And even after the infant seems to lose all interest in patterns *per se,* the patterning produced by the form, texture, fine details, and contouring of objects, surfaces, and people must be attended to for adaptive responses[17]; it is to be presumed that this is made possible in part by the early intensive examination of simpler varieties of patterning. That an infant or a monkey ignores the patterns on stimulus cards does not mean that a pattern denoting a feelable texture, a movable object, or a walkable surface will be similarly ignored.

Eventually, a further stage of development is reached in which patterning unrelated to spatial orientation or object manipulation is again a focus of attention. For monkeys, this stage may occur only after intensive discrimination training–training not of pattern-vision capacities but of pattern selectivities–to counteract the earlier learning to ignore such stimulus variations as irrelevant for everyday behavior. But in children, a similar change occurs, apparently without training or even encouragement. The young child develops an interest in picture books, photographs, representational drawings, abstract designs and scribbles, and eventually printed language. (This is not intended to deny the complexities of learning to read, but merely to suggest that the untrained interest in patterns on paper may be one essential predisposing factor.) This is a new stage of information processing, rather than a reversion to the earlier visual exploration of patterns, for the child is now much more efficient and retentive in his explorations and gives meaning to much of the informational content of the patterns. Furthermore, the motor skills and interests developed in the preceding stage can have a facilitating effect, as in the scribbling and drawing of the child, as suggested by Gibson[17]: "But scribbling is not simply play, . . . it is an opportunity for the educating of visual attention and for learning to perceive in new ways" (p. 230). Gibson has also cogently analyzed the perception of pictorial representations as distinct from and secondary to the perception of real objects and surfaces, the former being more difficult and developing later than the latter. (In this light it would not be surprising if the young infant failed to recognize a schematic or photographic representation of a human face, as suggested above.) How the perception of pictures and symbols on paper develops is not well understood. The research of Eleanor Gibson and associates[16] on the differentiation and attention to distinctive features of letters or other graphemes has given some clues to this process and illustrated some of the direct experimental approaches being applied. The improvement in discriminative capacities is clearly part of this developmental process; but alterations and refinements in visual selectivity are also essential for the exercising and use of these capacities. Changes in visual selectivity may in fact be of more critical importance, in that the basic oculomotor coordinations, visual acuities, and pattern-discrimination capacities are present long before they are put to use in deciphering markings on paper. And if the new visual selectivities of the young child are necessary for facilitation of the normal development of pictorial perception and reading, then aberrations in the development of these selectivities could easily cause retarded development of these complex perceptual achievements; such a possibility is at least worth exploring.

A conclusion of the recent research on infant perception, of perhaps more significance than the early presence of pattern-vision capacities, is the strong influence of selective attention to patterns in general and among various patterns during the earliest stage of perceptual development. The specific selectivities and the way they affect the development process change considerably, but the importance of their influence probably remains throughout all succeeding stages.

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LEWIS P. LIPSITT

Pattern Perception and Information Seeking in Early Infancy

This report concerns some of the research conducted at the Brown University Child Psychology Laboratories on infant learning. It may have some bearing on problems of reading disability, but in any event it is related to visual information processing and early infant experience, which I take to be the primary concerns of this conference.

TECHNIQUE OF STUDY

Fantz devised a most ingenious technique for determining what infants prefer to look at and, by inference, what they can see. His technique is so simple as to pose an embarrassment for the history of the child-development field; one would think that it might have been capitalized on before. Previously, people assessed the visual capacities of infants in terms of what they grasped with their hands. As you know, young infants do not grasp for very much, so that early investigators were inclined to assume that newborn infants do not see anything. Now we know better. Newborn infants do indeed see, and they profit from, or appreciate, and process visual information in important ways–i.e., their memories are affected by what they look at. Fantz's experiments, as well as others, indicate that newborn infants fixate objects and patterns in their environment with their eyes, and they fixate some objects and patterns more often or for longer periods than others. One can tell from the choice of one pattern or figure over another, or from the infant's responses to different visual patterns, what he prefers to look at, or at least what he discriminates.

A study done by Miranda (a former student of Fantz) has recently shown that even the premature infant can see patterned stimulation.[2] Miranda's study shows that conceptional age, in addition to actual age (age since birth), is a determinant of visual selection. To demonstrate that, he compared 27 3½-day-old, normal full-term babies with 27 premature infants, testing the latter group at less than 38 weeks of conceptional age. That is, the premature babies were tested, on the average, approximately 1 month before they would have been born as full-term infants. The stimuli were always presented in pairs, and Fantz's technique (which apparently has high reliability) involves observing whether the infant's eyes go to the left or the right. He used all the necessary counterbalancing of left and right figures to preclude erroneous inferences from position preferences.

PREMATURE INFANTS

The easiest way to summarize the Miranda data, without distorting the essential results, is to say that 1 month before the expected date of birth, but 3 weeks after birth, the premature babies' pattern preferences were essentially consonant with the visual-fixation behavior displayed by the full-term newborn babies tested at a mean age of 3½ days. In general, both the premature and the full-term babies resolved grating stimuli of 1/2 in. and 1/4 in.; they fixated these patterns more frequently and for longer periods than they did a plain gray stimulus. Both the premature and the full-term infants, moreover, tended to prefer the less complex of paired stimuli, but more consistently so for the premature babies of lower conceptional age.

FIRST YEAR OF LIFE

Fantz reports, on the basis of longitudinal data, that there is a marked change in discriminative capacity and in preference with age. He is conducting a longitudinal study from the ages of 2 to 52 weeks. Fantz does not posit many explanations for the marked change in preferences that infants display over that period, but he does suggest the possibility that experience is a primary consideration in determining what the child attends to most. He talks, for example, about the attention and interest value of various stimuli, which, according to his findings, seem to change drastically over the first year of life. The general finding within this period is that a plain, unmoving, unpatterned surface or object of any color or brightness is low in attention value at all ages. Younger infants in particular seem to like patterns–i.e., they attend to them more–and especially black-and-white patterns with well-demarcated boundaries.

It is also possible to conclude from the longitudinal study that by the age of 30–60 days a circular figure, such as a bull's-eye, is preferred over a linear figure. That is the age range that Dr. Kagan has described within which some remarkable changes occur (see p. 324). The schematic face seems to be preferred at birth over a picture of a real face. The schematic face is fixated more than a photograph of a real face until 16 weeks of age, when the child comes to fixate more on the real-life likeness. By 20 weeks of age, the child prefers a schematic face over a jumbled face. A solid face (three-dimensional) is preferred with increasing age over two-dimensional figures, and markedly so by 8 weeks of age. By 6 weeks of age, an unfamiliar figure tends to be preferred over a repeatedly exposed figure.

HABITUATION

I hope that Dr. Kagan's presentation does not leave the impression that habituation and learning processes do not occur in the human infant until he is 30, 60, or 90 days old. A growing body of data indicates that newborn children are remarkable habituators to stimulation of many sorts. Some of the best data in the field are derived from olfactory-stimulus work, and much work has also been done with auditory stimulation.

In our own laboratory, we have done a series of experiments in which we delivered to the newborn child such olfactory stimuli as anise oil. The newborn child does indeed respond with a startle-like reaction, and when stimulation by these odorants is repeated, the response declines in even as few as 10 trials. The newborn habituates quickly to olfactory stimuli, as well as to other stimuli that we administer in the laboratory. Figure 1 shows a visually aware and alert child being stimulated with an odorant on the end of a swab. The infant's response includes respiratory disruption, heart-rate acceleration, and bodily movement measured on a stabilimeter. These infants, all within the first 4 days of life, are hooked up to a polygraph from which we record a number of different behaviors. We are also doing habituation experiments with the sucking response. The babies suck an artificial nipple that contains a device permitting us to record their rate and intensity of sucking. Presentation of a tone or odorant interrupts a baby's sucking; successive presentations of the same stimulus will produce a diminishing amount of interruption.

Once the newborn child is habituated to a given odorant or tone, presentation of a different stimulus without violation of the temporal

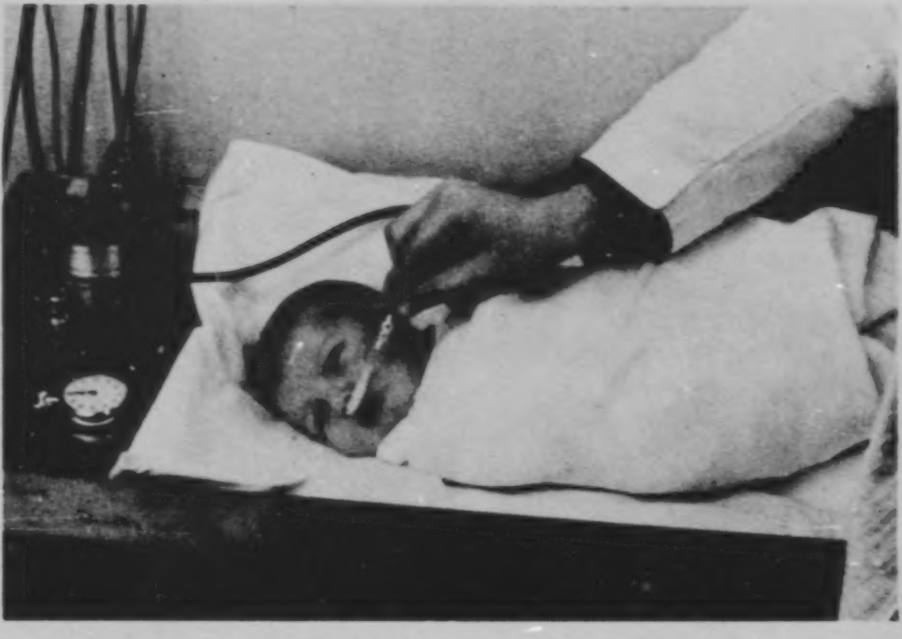

FIGURE 1 Awake and alert newborn being administered an odorant on cotton swab.

sequencing of the stimuli will cause the response to recover—i.e., dishabituation will take place. The newborn can tell us which odorants he is sensitive to both by his initial reactions to stimuli and by his recovery behavior after habituation to stimuli. Thus, the infant "reports" to us when he can discriminate the difference between two stimuli. For example, if he habituates within 10 trials to the anise oil, and then we change to asafetida, his response will recover and he will thereby tell us that he noted the difference between asafetida and anise oil. (Strength differences of the odorants are controlled through counterbalancing.)

In an elaboration of this procedure, we mixed odorants (A and B) and delivered the mixture to the infants. After habituation, we administered A or B separately in the same diluent. In this situation, the child similarly dishabituates, telling us that he can discriminate between odorant A or B and the mixture of odorants A and B.

We think that we are dealing here, not with a peripheral phenomenon, but rather with a central nervous system function, or sensory integration. We think that the last type of study, in which we induce habituation to mixtures and then administer single components of those mixtures, brings us closer and closer to a learning process.

REINFORCEMENT

Kagan's inclination, I think, is toward the view that a child does not become a habituator or learner until perhaps 30–60 days of age. Studies of newborn children show that they learn; the best evidence comes from a series of studies in which Dr. Einar R. Siqueland and I have potentiated the sucking response through reinforcing circumstances. Stimulating a newborn child on one side of his mouth elicits an ipsilateral rooting reflex.

Head-Turning

I am getting into head-turning behavior now, which I think is close to eye-turning behavior, which in turn should be related to reading behavior.

In Figure 2, it can be seen that the baby's eyes are open and that he has a rather alert appearance. Infants while sucking do not always go to sleep, as we so often read. We hear all about how pacification produces

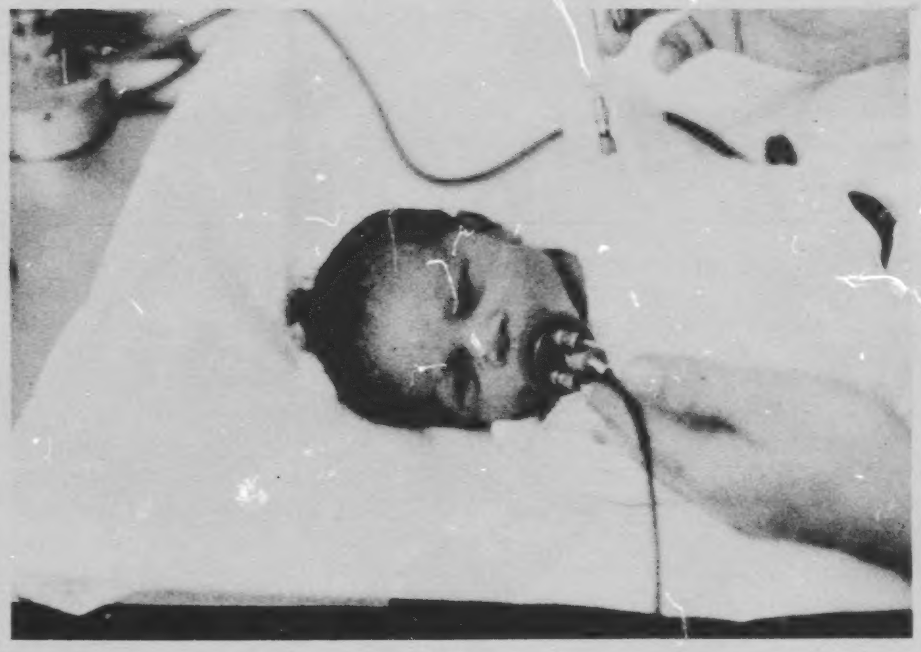

FIGURE 2 Newborn infant sucking on automatic nipple device that enables polygraphic recording of suction. Three wells surrounding the transducer enable administration of different nutrients or tastes.

sleep, a general lessening in body tone, and so on, but if the child has recently slept and is now feeding, putting a nipple in his mouth will probably arouse him. It arouses him visually and it arouses him with respect to head-turning activity. Many mothers report that they are able to communicate with their newborn babies best while they are feeding them. The child looks up at its mother's eyes, and while sucking looks around at the corners of the room and at other interesting objects in the environment. We ourselves have not studied the looking behavior of the young child, but we have been observing other response processes, such as head-turning behavior, and I think that head-turning and eye-turning responses are innately related. The situation pictured in Figure 2, incidentally, is one in which we are able to record the child's sucking characteristics polygraphically. I might say as an aside that we also have the opportunity, using this automated technique,[1] of feeding the infant as a consequence of certain types of behavior. We can reinforce him through wells that enable the introduction into his mouth of dextrose solutions or milk in controlled amounts, not only to compare the relative attractiveness of gustatory stimulation, but also to reinforce the child in learning studies.

Conditioning

In our conditioned-head-turning studies, we capitalize on a response that is in the newborn child's repertoire of congenital responses: the rooting reflex or the turning of the head to the stimulated side. We feed the child when he turns his head after we touch him on the face. The touch induces a head-turning response about 25% of the time. If we sound one tone, touch the baby on the left side of his face, and feed him for turning there, the head-turning response in that direction goes up from 25% to 75%. In the same child and on alternate trials, if we sound another tone and touch in the same place but do not feed him for turning his head (i.e., do not operantly reinforce the response), the frequency of the response will either remain the same or even go down to around 20%. We are thus able to establish discrimination in the newborn child on the basis of tones and touch to the cheeks, feeding differentially in the presence of the two tones.

An elaboration of that technique involves switching tones A and B. If A was the previously reinforced tone, such that the child was fed for responding to tone A plus touch and has come to respond 75% of the time, we can make A the negative tone and B the positive tone, and we get flip-over behavior. The child who was previously responding more to A than to B (A having been reinforced) can now be made to respond more to B than to A simply through changing the reinforcement contingencies.[4] Even the newborn child, not to mention the 2-week-old or 2-month-old child, is remarkably receptive, and his behavior changes markedly in consequence of the stimulation provided.

In speaking to nurses with considerable experience with newborn babies, I have often been amazed, in view of their extensive opportunities to observe infants, that many do not believe that newborn babies can see. When we invite them into our laboratory and show them newborn babies' horizontal and vertical scanning of dangling objects, they are often overwhelmed and begin to wonder about their own visual perception.

In addition to our newborn-infant laboratory at the Providence Lying-In Hospital, we have a research facility at St. Vincent's Home for Infants, an orphanage in Rhode Island. Figure 3 shows an apparatus for head-turning experiments at the orphanage. The head-turning is recorded on a polygraph, so that it is possible to note its direction and extent. The apparatus would be suitable for the study of visual exploration in infants, although we have not used it that way. When we went to work in the infant home, we discovered that the nuns had suspended mobiles over the cribs of infants as young as 5 days of age. That was not part of our experiment; it just happened to be what was (and is) done in that particular institution. The personnel were aware (before we arrived on the scene) of the literature relating to effects of stimulus deprivation upon the development of personality and other psychologic attributes.

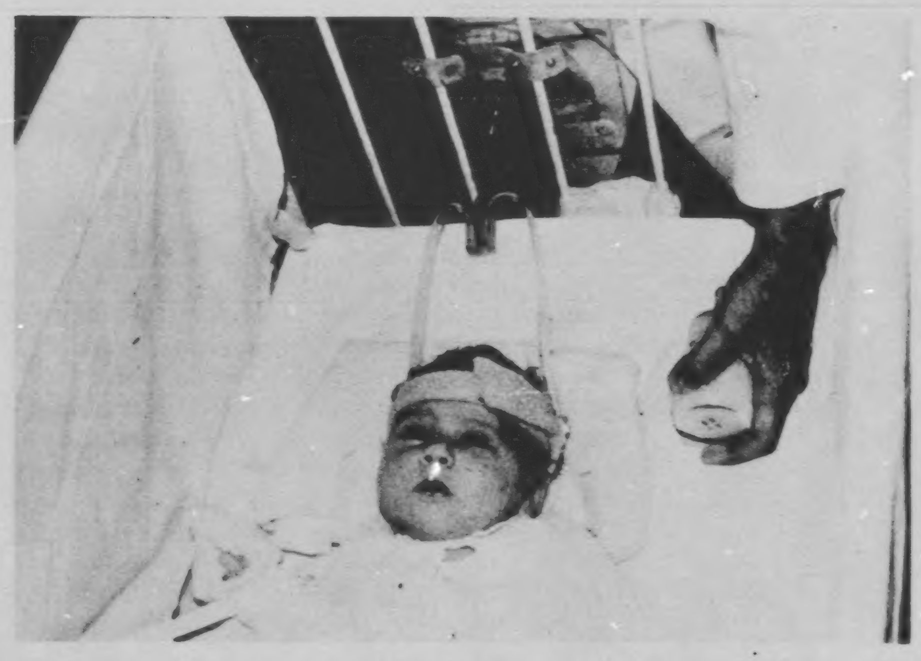

FIGURE 3 Infant attached to apparatus for recording head-turning. The plastic yoke is attached to a rotary potentiometer allowing polygraphic recording of incidence and magnitude of head-turning activity.

Conjugate Reinforcing with Mobiles

Stimulated by the presence of the mobiles there and by some suggestions of J. McV. Hunt[5] concerning their attractiveness for infants, we did some studies with mobiles. There is a vast amount of literature on operant conditioning in animals and children. Most of the work has used what I would call pellet reinforcement–the delivery of something that is discrete and can be picked up by the child or popped into his mouth, such as a small piece of candy. But concentration on that type of reinforcement reflects a restricted view of what reinforcement is like in real life. Picture, for example, what goes on between a mother and a nursing child. Whatever reinforcement occurs is not of the pellet variety. When the child is sucking at the breast or bottle, the harder he sucks, the more food he gets; reinforcement is commensurate with the frequency and intensity of the sucking. I submit that visual reinforcement in real life is more like that, and not so much like pellet reinforcement. We implemented research involving visual reinforcement of the hanging-mobile type, in which the child controls his own visual input from his surroundings. Now, most parents and nurseries simply suspend the mobiles above the crib. What would happen if, instead of suspending the visual stimulation before the child, we enabled the child to control his own input? That is really what we all do when we turn our heads and move our eyes. We control our own visual input, presumably in some sort of rational manner having to do with the curiosity or interest value of the stimulus.

Carolyn Kent Rovee, a former student in the Brown University Child Laboratories, implemented a study with her own 2-month-old child and with five other children of similar age, using a type of reinforcement such as that just described.[3] It is called "conjugate reinforcement"; I think Ogden Lindsley devised the term to signify situations in which the subject is in direct control of the reinforcer, the reinforcer occurring immediately and being commensurate in intensity with the activity of the subject. Pressing on an accelerator pedal would be an example of this sort of interaction between a subject and his environment; turning a spigot to obtain water, in greater amounts with increasing turns of the spigot, would be another example. Two observers recorded the responses. They obtained baseline measurements of activity of, for example, the infants' right arms; if an arm moved a specified minimal distance, that would be called a response. Each baby's right arm was

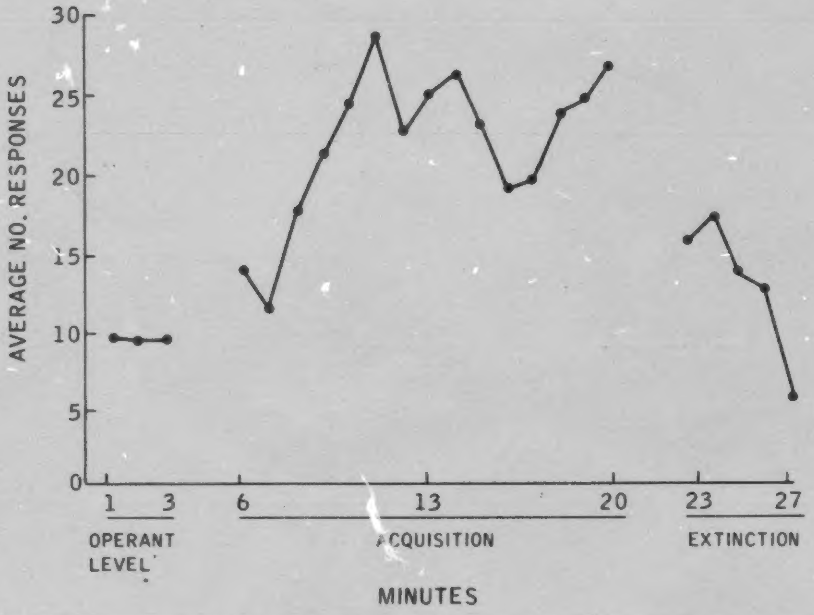

FIGURE 4 Mean rate of response as a function of conjugate reinforcement condition in 18 infants 9–12 weeks old; leg attached to mobile during acquisition. Each point represents a successive minute of observation. Each curve represents six subjects. Modified from Rovee and Rovee.[3]

then tied to the mobile hanging above the crib, so that when the baby moved its arm, the mobile moved.

Figure 4 shows some data from Dr. Rovee's experiment. First, an operant level may be seen, consisting simply of a 3-min period, during which she recorded the number of responses from each of the six children while they were in the presence of the mobile but not operating it. This was followed by a 15-min conjugate reinforcement phase. The effect of this reinforcement on the babies' activity levels was highly significant. Dr. Rovee later introduced an extinction period by detaching the cords from the babies' limbs. During extinction, there was a diminution of behavior, and the children gradually became uninterested, or at least less interested in the mobile than they had been before. The figure shows a cumulative recording of a full minute of the limb activity of a child; the child has had a minute to become higher in level of response to the manipulable mobile than during baseline measurement, when responses did not affect movement of the mobile. By the time the first minute of the conditioning phase is over, the infant has already had an opportunity to learn that his actions control the mobile. However, there is not a reliable difference between the last point in baseline and the first point in conditioning.

The increase in activity between baseline and conditioning over minutes of recording is highly significant. The infant is learning to control the stimulus. Then, in extinction, he learns that he no longer controls it. There is actually a very fast dropoff in activity when the baby's behavior no longer has anything to do with the control of the visual stimulation. This is also a reliable difference between the conditioning and the extinction phases. Figure 5 deals with a possible objection to a learning interpretation of the effects shown in the previous figure. It might be argued, for example, that the effect obtained is only apparently a learning phenomenon and that it is due to a baby's becoming

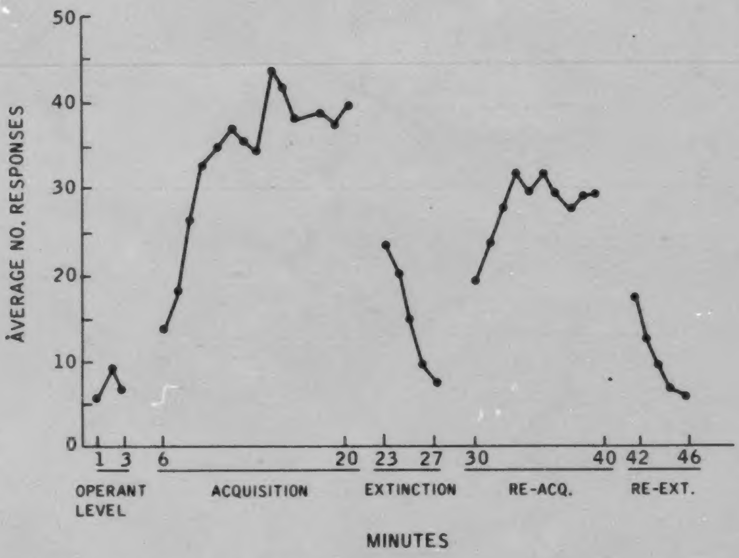

FIGURE 5 Mean response rate as a function of conjugate reinforcement condition over 46 min of continuous observation in four infants from previous group; leg attached to mobile during acquisition. Modified from Rovee and Rovee.[3]

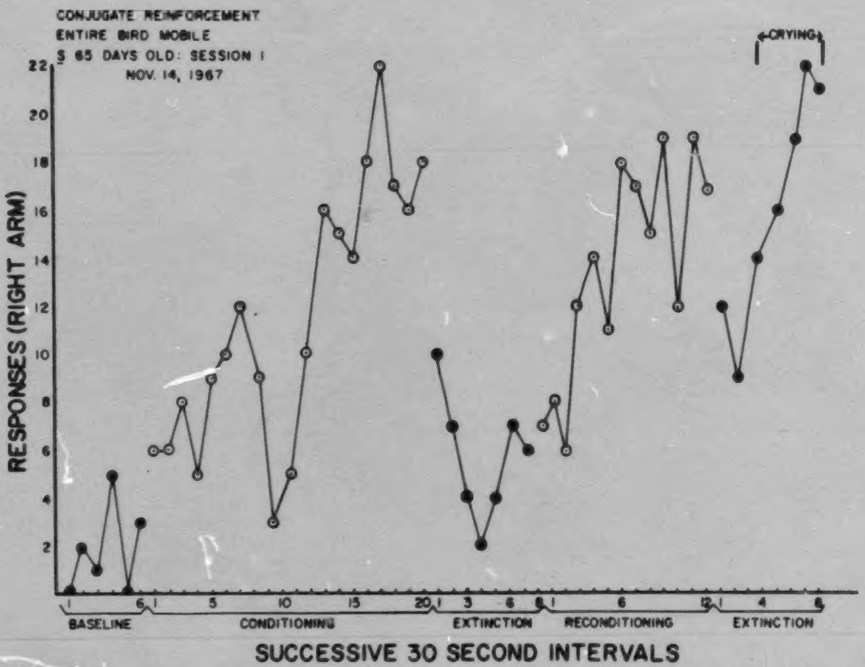

FIGURE 6 Unpublished data of Smith and Lipsitt showing operant responding of infant in conjugate mobile situation. During baseline, infant's right arm was not in mechanical contact with mobile. During conditioning phase, the cord was attached between the baby's arm and the mobile, following which the cord was again detached for an extinction session. Note that the onset of crying elevated responses in the second extinction session.

increasingly alert, with exposure, to the mobile (and perhaps to the experimenter), and that the eventual diminution of behavior during extinction is really a fatigue effect. But Rovee was able to keep four of the six subjects in a study of reacquisition after the extinction phase. There was recovery of the conjugate reinforcement effect, and if the subject's limb was released from control of the mobile, his activity then declined.

That the infants could be shown to intensify their behavior when in control of their own stimulus input and to reduce their behavior when the response was no longer pertinent to visual input indicates that lively visual stimulation may serve as a reinforcer to enhance learning behavior. It might also be speculated, conversely, that the infant's opportunity to control his own visual environment enhances the attractiveness of that stimulation.

Following Dr. Rovee's study, I was fortunate to have another student, Mrs. Leslie Smith, who decided to follow her own baby over a longer period in this type of mobile reinforcement situation. Her data relate to the behavior of this one child successively tested over several months and with different mobiles. The responses of this baby, first seen at 6 weeks of age, may be seen in Figures 6 and 7 in the baseline condition. Following this, Mrs. Smith attached the child's arm or leg to the mobile in a manner similar to the procedure used by Dr. Rovee. Mrs. Smith, however, introduced an automated procedure for counting responses; the lead to the counter is attached to the baby's limb along with the mobile, so that a minimal movement would activate the re-

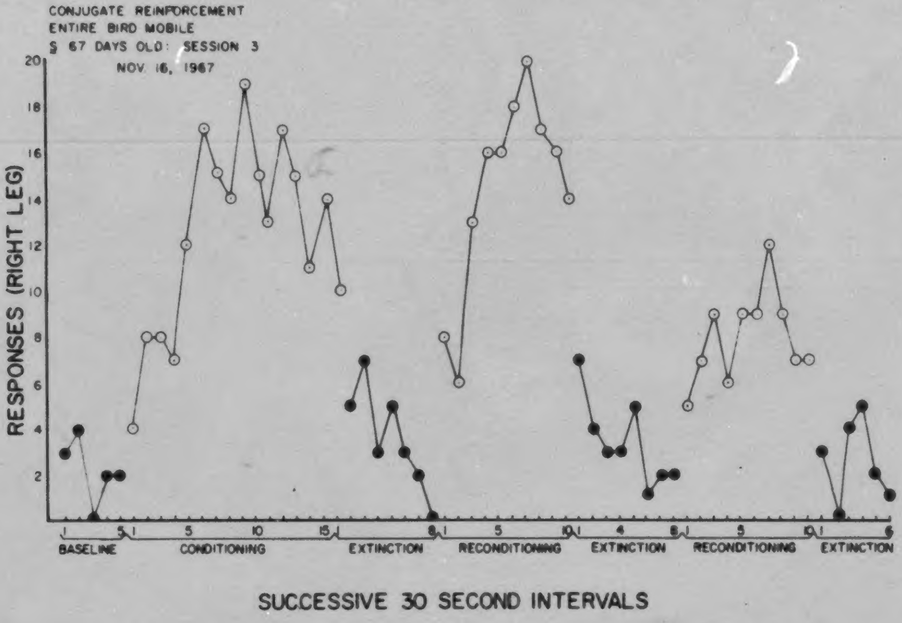

FIGURE 7 Unpublished data of Smith and Lipsitt. In third training session, infant's right leg responses activated the mobile during conditioning periods. Response is elevated during conditioning, in contrast with performance during baseline and extinction periods.

corder. It may be seen that when the mobile was controlled by the child there was an increase in response, then a decline under extinction conditions, then an increase and decline again, and so on. The conjugate reinforcement phenomenon obviously occurs even at 6 weeks of age (rather, it can occur; there are undoubtedly individual differences about which we know very little), and it can be a striking phenomenon. Note that in this extinction phase the baby began crying, following training in control of the stimulus, when his control of it was removed. This crying phenomenon occurred frequently in this child during extinction phases and Mrs. Smith responded, as would most mothers, by making the mobile's movement once again available to the child, whereupon crying stopped. This phenomenon may well be akin to or a precursor of what is called "separation anxiety" in older children. When Mrs. Smith changed the mobile, she found that introduction of a novel mobile enhanced the response of the child. On the basis of data obtained later, it seems to us that this was not merely an effect of novelty, which was systematically checked out, but was a true preference of a red mobile over a white one (Figure 8). Again, response level went down sharply during extinction, although the child was still in the presence of the red mobile.

Conjugate Reinforcing with Sucking

We are also seeing infants at various ages in the Hunter Laboratory for conjugate reinforcement experiments devised and conducted by Professor Siqueland. In these experiments, the child controls his visual input through his sucking behavior. The visual stimulation is presented on the screen (Figure 9). The more frequently and more intensively the baby sucks, the more visual stimulation he gets, because the projector is equipped to provide greater illumination with greater intensity or frequency of sucking. It can be set in such a way that the child's visual stimulation will be proportional to both the rate and the intensity of sucking. One can use a psychophysical-threshold procedure, whereby the child has to suck with a certain intensity or the projector is not triggered. In such instances, one can operantly reinforce only high-intensity sucks, and not low-intensity sucks. From the behavior of control subjects in this situation, it appears that high-amplitude sucking will wane over time if it is not reinforced. Thus, the control group shows a decrement in sucking behavior relative to the group whose behavior produces increased visual stimulation. The parallel between this technique and the conjugate mobile technique is perhaps obvious.

Figure 10 presents data from three groups of 10 4-month-old children in one of Dr. Siqueland's studies. One group had its sucking behavior reinforced with light onset and light intensification. Another group was a baseline group, which received no visual stimulation for sucking; the babies had pacifiers in their mouths and their sucking behavior was recorded, but the sucking did not control the visual feedback. A third group, represented by the dashed line that begins at the bottom, got reinforcement withdrawal with increases in sucking; that is, this group started with the visual stimulus already full on, and successive or repetitive sucking on the device produced withdrawal or

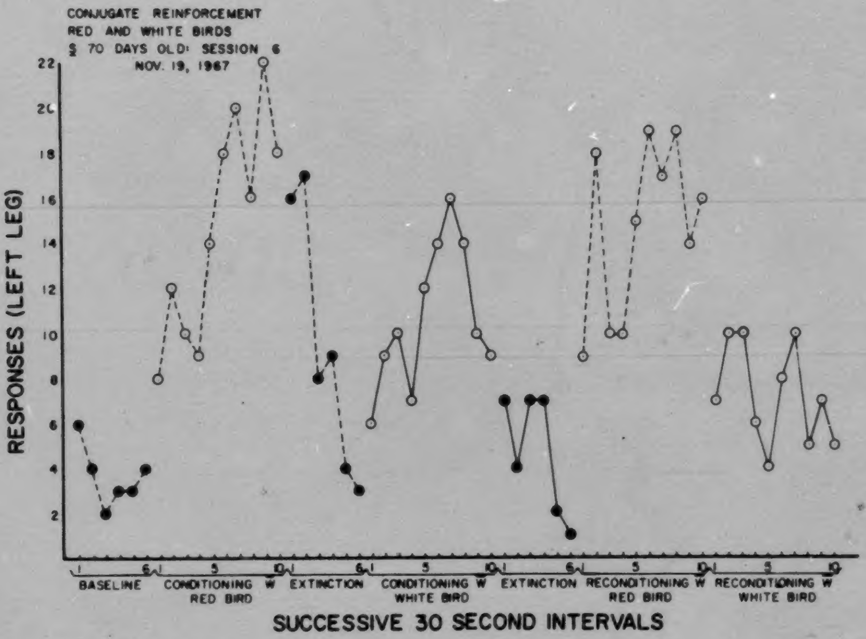

FIGURE 8 Unpublished data of Smith and Lipsitt. Infant's responses to two mobiles, one red and one white, are compared in successive sessions. Response levels tend to be higher during presentations of the red stimulus than the white stimulus, and both tend to be higher than performance during extinction conditions.

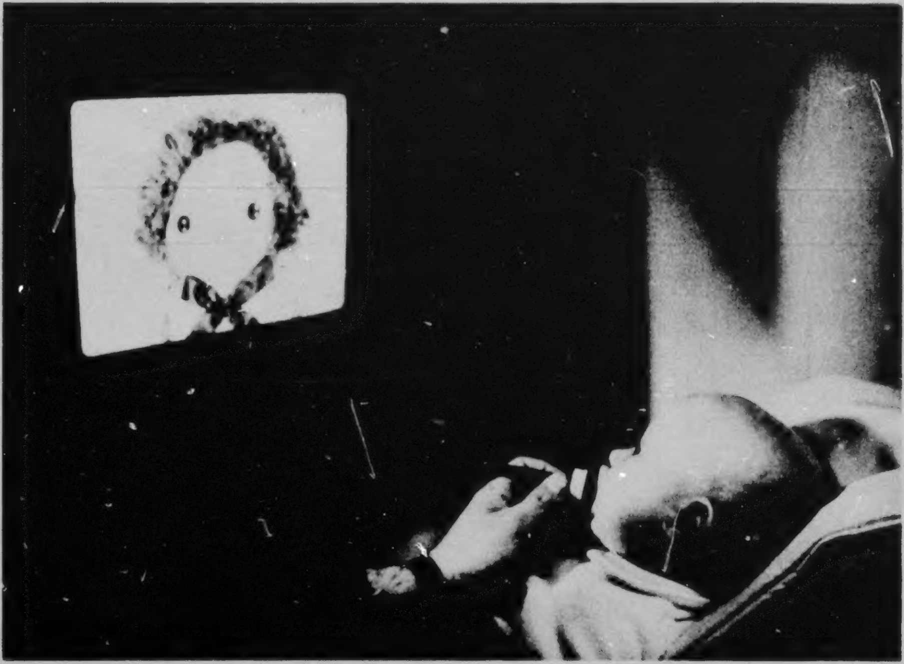

FIGURE 9 Infant operating sucking manipulandum. High-intensity sucks increase illumination of slides shown on screen in front of subject. (After E. R. Siqueland.)

diminution of illumination of the visual stimulus commensurate with the high-amplitude sucking.

These children, 4 months old, were being operantly reinforced for their sucking behavior, and the behavior was controlled by the "novelty" and attractiveness of the stimulus. This technique should enable us to increase our understanding, as did Fantz's innovations, of attentional processes in the infant and the preferences that infants have for different stimulation at different ages.

IMPLICATIONS IN PREMATURITY

We will be able to follow some of these children when they become readers and perhaps see how some of our intervention procedures do work. For example, we have a study in our Child Study Center with premature children, who are fortuitously subjected to a relatively impoverished environment, sometimes for as long as the first two months of their lives. The natural state of affairs in the ordinary premature nursery of most hospitals is such that the infants exist in what may be a rather deprived setting compared with the experiences that normal

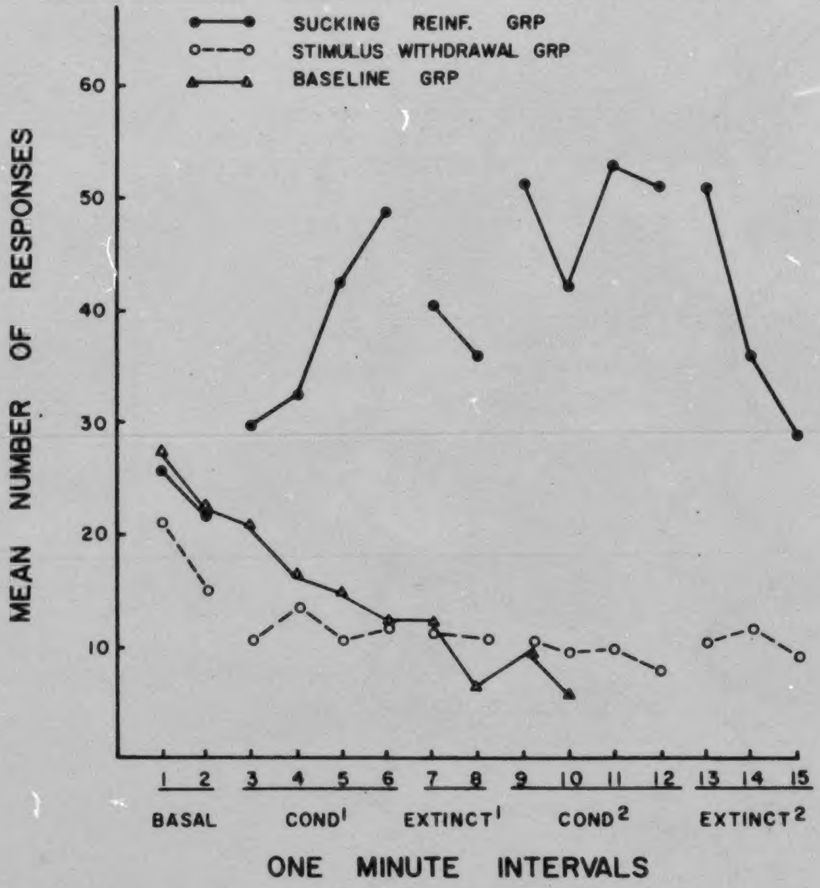

FIGURE 10 Unpublished data from experiment of Siqueland involving three groups of 10 4-month-old infants in whom sucking activity produced different visual consequences. One group received visual reinforcement; the sucking behavior of a second group was associated with stimulus diminution; and a third group served as a control, with no visual changes occurring during the recording of sucking behavior.

full-term newborn infants have outside an incubator, in contact with adults. There are considerable data available to indicate that the premature child is in developmental jeopardy for some time after he is discharged from the premature nursery and is growing up normally. Available documentation indicates that premature children as a group tend to have lower intelligence, be poorer readers, have more difficulty in school, and so on. Statistically speaking, the premature child has handicaps that seem to persist, and one has to wonder why. Some biologic explanations might be invoked: the viability of the organism was in jeopardy from the start, it suffered respiratory distress, and it did not have the full complement of fetal nurture normally received by the full-term infant. But one has to entertain an alternative hypothesis, namely that part of the deficit results from the understimulation that is made necessary for the first few weeks or months of postnatal life by the biologic prematurity. Dr. Siqueland is attempting to provide compensatory stimulus enrichment to premature babies through various means. The children are reinforced for looking, and they get picked up, rocked, and spoken to by motherly nurses; in this way, they are provided with extra visual and other stimulation of a wide variety. It is the intent to follow up these infants for a long period to find out what effects occur over the long run, in contrast with a control group that receives the routine premature care.

On follow-up, one of the situations to which the infants are subjected is the sucking visual reinforcement previously described. Thus far, a number of children have come back at 4 months of chronologic age, some of them having been out of the hospital for only a couple of months; among these infants are several sets of twins. When the stimulated twins are now tested in the controlled learning situation, those who have had such training seem on the basis of preliminary findings to learn faster in this conjugate reinforcement situation. The stimulated and nonstimulated premature babies are tested "blind" by an assistant who does not know whether they are stimulated or control subjects. First indications are that at least some learning effects are facilitated by early stimulation of the premature children.

RELEVANCE TO READING

Let me try to draw some conclusions in an attempt to make these data and this program of studies relevant to the concerns of the present conference. It should be apparent that very young infants do indeed learn. It should also be obvious from our conjugate reinforcement data that infants are captivated by visual stimulation, particularly if it is novel. It is important to note, however, that some of our most striking learning effects in infants appear in situations in which the baby himself is acting on the environment to change his visual input.

What does all this mean for reading disability? First, the human being is very responsive to stimulation, including visual stimulation, from birth onward, and techniques are now available for extensive and longitudinal study of the ways in which newborn and older infants are affected by their visual surroundings. Second, it is now inappropriate for us (educators and psychologists alike) to think in terms of merely waiting for maturation of some behavioral processes. We now know that the failure of some kinds of behavior to occur is experientially induced, and it appears that the overemphasis on maturational processes as primary determinants of development has, to some extent, precluded or inhibited our full exploration of the real learning potential of children at different ages. Too often we are inclined to say, when a child does not seem to be learning to read well, that he is not mature enough, that he is not "ready" for reading. That position is often just an escape—a poor excuse for our lack of competence in devising appropriate techniques for producing the desired behavior. We child developmentalists and educators cannot retreat much longer into the maturation argument to account for our failures to implement appropriate techniques to facilitate education in children of diverse experience.

We saw from the presentation of Dr. Flom (p. 291) that by 3 years of age at most, and I would guess by 1 or 1½ years (if not before), the child has the appropriate visual-acuity equipment to do a lot of things that were at one time thought impossible for the very young. The argument that 4-year-olds, for example, lack the appropriate visual apparatus to read has obviously been refuted, but that is exactly the kind of "reading-readiness" argument that has been offered in the past to forbid earlier exploration of children's reading potential. I do not advocate teaching 1-year-olds to read, and in fact I do not know whether it is possible. Moreover, I have no idea as to whether it would be *good* to teach a 1-year-old or even a 3-year-old to read; that is quite another issue. But because the early indications are that it is possible to do so, we can no longer resort to the often-voiced argument that it is better to wait for maturation of "reading readiness" to evolve. From the scientific point of view, moreover, the learning components of "reading readiness" should be given further study. Certainly, perceptual pretraining and transfer of the effects of such training to the reading situation constitute important areas for exploration. Pretraining with visual stimulation of the sort that I have described here, perhaps along with coordinated auditory stimulation such as occurs in real life, must be exceedingly important in the development of fascination with reading materials on the part of children who read well and who like to read.

It is my belief that children who read well and who read a lot find some sort of joy in reading–joy in controlling their own environment–very much as the children in the conjugate reinforcement situation seem to enjoy manipulating their own visual input. Controlling one's own visual environment (and hence perhaps one's visual fantasies and imagery) must be one of the reinforcing characteristics involved in the persistence of reading behavior. If a child is going to enjoy reading, or enjoy the "ingredients" or precursors of reading–such as looking at books around his home, looking at the printed word, and looking at pictures–he has to be reinforced. That is, for curiosity behavior to be perpetuated, it seems to me that some sort of reinforcement process is involved in looking at things in the environment, the kinds of things that reading teachers get children to observe before they are presented with the printed word.

My point–and perhaps it is far-fetched–is that the kinds of processes we are dealing with in this research on infants capitalize on the curiosities of the child with respect to visual stimulation and reinforce the child for looking, or for being curious. I think that this is the sort of thing that occurs in the natural process of growing up and that reinforcement for looking constitutes a precursor for reading behavior. For example, if you punish a child every time he looks at a printed page, he will soon not have much joy in looking at the printed page, and that is probably what some teachers actually do to children during the early phases of reading training.

We should note that early readers are good readers. It is possible that both attributes are simply correlates of intelligence and that intelligence is really the primary cause of both attributes; but I think that it is reasonable to consider the possibility that early visual and other stimulation is an important determinant of all three–early reading, proficient reading, and intelligence.

Finally, I think that reading disorders call for developmental investigations of learning processes and learning disorders. The antecedents of reading disabilities must have a good deal in common with other developmental and learning attributes. We will know more about developmental processes generally when the learning of reading is better understood, and we will know more about reading aberrations when developmental and learning processes are better understood. Similarly, learning disorders call for the discovery of new types of remedial techniques involving learning principles. Even children who have true brain dysfunction have to learn and do learn new modes of behavior. A diagnosis of brain dysfunction does not preclude the beneficial administration of compensatory training. It is certainly inappropriate to assume that, if a child has central nervous system anomalies, there is nothing for the learning psychologist or the educator to do about it, from either the scientific or the remedial standpoint. Appropriate reading techniques are necessary for the dyslexic child no less than for the neurologically intact child.

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THE ROLE OF INFORMATION PROCESSING IN PERCEPTUAL AND READING DISABILITIES

THOMAS T. S. INGRAM

The Nature of Dyslexia

In this paper, I shall attempt to describe what has been and is meant by the terms "dyslexia," "specific dyslexia," and "developmental dyslexia"; these are common terms that have become confusing because authors have used them in so many different ways. I shall discuss a number of clinical syndromes involving serious difficulties experienced by apparently healthy children of average intelligence in learning to read and write. Some relevant work concerned with difficulties in learning to read and write carried out in the Department of Child Life and Health in the University of Edinburgh will be described.

Reading and spelling disabilities acquired as a result of disease of the brain occurring when reading and writing skills have been developed will not be discussed.

HISTORICAL REVIEW

Many differences of opinion about reading disability in children have their origin in the ideas of earlier authors and in misconceptions about their work.[21] At the risk of reviewing much literature that has already been discussed extensively, it seems worthwhile to describe the ways in which specific difficulties in learning to read and spell came to be recognized. In the process, many terms were used without agreement on their definitions.

The Recognition of "Word Blindness"

In the last half of the nineteenth century, neurologists were particularly concerned with allocating specific psychologic functions to specific areas of the brain. The identification of a "speech center" by Broca[14] was followed by efforts to define parts of the brain that were particularly concerned with reading and writing. Descriptions of adult patients who lost the power to read and write as a result of a variety of brain lesions were soon published.[4,5,7,13,84] Henschen[57] attributed word blindness, usually accompanied by difficulty in writing, to a lesion of the left angular gyrus. Déjèrine[30] considered that the lesion responsible for "alexia" was in the medial and inferior portions of the left occipital lobe.[12,21]

At the same time as the first papers on "congenital word deafness" and "developmental aphasia" or "congenital aphasia" were being written, the first inadequate accounts of children who were unable to recognize written words or letters appeared. They were considered to suffer from "congenital word blindness."[78,100] Soon, further reports described otherwise healthy children of apparently average intelligence who were "word blind" or "letter blind." Most of the early reports were by ophthalmologists.[48,64,66,122,126] It was perhaps inevitable that, just as children who suffered from "congenital aphasia" were assumed to have brain lesions similar to those found in adults suffering from aphasia, so children with "congenital word blindness" or "congenital alexia" were suspected of having lesions similar to those described in adults who had lost the power to read and write. Fisher,[49] for example, suggested that congenital aplasia of one or both angular gyri might be responsible for "congenital word-blindness." It was difficult, however, to reconcile that interpretation with reports of quite large numbers of familial cases of "word-blindness" that soon appeared.[48,117,122,126,131] A family in which six cases occurred in two generations, including four in one sibship of 11 children, was described by Hinshelwood in 1917.[65]

As Critchley remarks,[21] "what might be called the early history of this condition [developmental dyslexia] was closed by 1917, when Hinshelwood brought out his second monograph entitled *Congenital*

Word-Blindness" (in which he reviewed his experience with 31 cases of congenital word blindness and summarized the work of other authors up to that time). Hinshelwood defined the condition as

> a congenital defect occurring in children with otherwise normal and undamaged brains, characterized by a disability in learning to read so great that it is manifestly due to a pathological condition and where the attempts to teach the child by ordinary methods have completely failed.

Congenital word blindness occurred more commonly in boys than in girls and was often hereditary. Some children had difficulties in identifying whole words but could identify letters; others had difficulties in identifying individual letters. Some, but not all, could not recognize written figures. Hinshelwood considered that the disorder was the result of a failure to develop the brain centers concerned with the visual memory of words, letters, or figures. Auditory memory was commonly unaffected. He made the acute observation that some children could read text if they were allowed to read aloud or even move their lips silently while reading, but could not read silently without movement of lips, tongue, or palate. Most patients could copy written material, but they were quite unable to write to dictation. Hinshelwood interpreted this as indicating that there was a deficiency in the ability to remember letter and word shapes. Because of their difficulties in reading, many children guessed at words that they could not identify and used accompanying pictures as clues to the contents of the written material. He noted that many patients who suffered from "congenital word blindness" improved as they grew older and responded to teaching.

Hinshelwood's account of the clinical findings in children who suffered from "congenital word blindness" was remarkably complete, although he did not comment on the frequent history of associated retarded speech development or on "crossed laterality," an increased tendency to left-handedness and ambidexterity already noted by other authors.[93-95] McCready postulated that there were common etiologic factors in "word-blindness," "congenital word-deafness," "delay in the acquisition of speech," and "stuttering." All these disorders could be regarded as being the result of "biological variations in the higher cerebral centres causing retardation" of the various functions of language. He thought that they were the result of variations in the degree of cerebral dominance that was also manifest in weakness of lateralization of handedness shown by many patients. Hinshelwood's idea that underdevelopment of cerebral dominance was important in determining the disturbance of spoken and written language in his patients probably had a considerable influence on later authors, particularly Orton.[103,106]

Study of Perceptual Defects

After Hinshelwood's work, further studies of patients with difficulties in learning to read and spell were devoted largely to more accurate descriptions and analyses of their difficulties in recognizing, orienting, interpreting, and reproducing written symbols of spoken language. Bronner,[15] in *The Psychology of Special Abilities and Disabilities,* appears to have been the first author to study systematically the nature of the perceptual defects associated with reading disability. She attributed reading disability to faulty visual memory in one child and to poor auditory discrimination and memory in another, but in many other patients she was unable to discover any evidence of perceptual deficits and was at a loss to account for the children's difficulties in learning to read. She speculated as to whether reading required some specific synthesizing ability that it was not possible to study with the means at her disposal. Fildes[47] observed that children who failed to learn to read and write had difficulties in recognizing and reproducing shapes, designs, and patterns other than those found in written material. Unfortunately, a high proportion of her patients were mentally defective; her findings may have been due partly to the fact that the discriminatory powers of her subjects were immature at the time of testing.[62,63] Gates[52] found that children aged 8 or more with reading disability were as able to detect small differences in the shapes of test objects as were those who had no reading difficulties. He denied that visual perception or memory could account for reading disability and thought unfavorable environmental factors–including defective teaching, poor home background, and emotional disturbances–were more important than perceptual difficulties in causing reading disability. Bachmann,[3] using tachistoscopic exposure techniques, came to similar conclusions. He was particularly impressed by the fact that children with specific reading difficulty read extremely slowly. Ombredane[102] found no abnormalities of visual form discrimination in three carefully studied children aged 12–14 who had reading difficulty.

The contemporary studies of Feyeux[46] have not received the attention they deserve. Not only did she describe accurately the findings in children with retardation of speech, but she also noted how frequently it was associated with difficulties in learning to read and write. She emphasized that reading and writing difficulties must be considered to be parts of a general language disability, although she recognized that a proportion of children with difficulties in learning to read and spell showed no obvious abnormalities in the development of speech.

Reversals Recognized

Orton[103,106] emphasized the extent to which children who suffered from "word blindness" tended to reverse the order of letters and syllables in words or of words in sentences. He noted the instability in recognizing and recalling the orientation of letters and the order of letters in words, which he called "strephosymbolia" (twisted symbols). In writing, this was manifest by the fact that letters were often malformed, apparently because the child was vague about the shape he was trying to reproduce. They were often reversed wholly or in part, and the order of letters, syllables, or words was often reversed or otherwise confused. Thus, spelling was very unreliable and inaccurate. Many patients could write and read mirrorwise much more easily than unaffected children.

Orton[105] was impressed by the frequency with which he found his patients to be ambidextrous or left-handed or to show conflicting laterality of eye and hand. He hypothesized that in his patients there was an intermixture of control in the areas in the two hemispheres of the brain that serve the visual or reading part of the language function and that in normal children are active only in the dominant hemisphere. He suggested that there might be conflict between the mirror images in the two hemispheres when word-blind children attempt to build associations between letters and spoken words. As a result, confusion of orientation and sequencing might occur:

> the data we have assembled from the study of left handedness and of various language difficulties in the family stock of children who have a specific disability in learning to read and show the strephosymbolia syndrome give what to me is convincing evidence that such children represent intergrades between right-sided and left-sided familial tendencies and that the reading disability follows fairly definite hereditary trends. . . . In families with this disturbance there are also more than the expected number of left-handed members and persons with delayed

> speech, stuttering, reading, writing and spelling disabilities, and abnormal clumsiness (developmental apraxia). In the childhood histories of children who come to attention as presenting reading and spelling problems we not infrequently find indications of developmental deviations in their acquisition of speech and motor patterns.

He was careful to point out that, although he had described "developmental word deafness," "developmental motor aphasia," "developmental alexia," and "developmental agraphia" separately, these disorders were in fact commonly found together in individual patients. He emphasized their common etiologic and clinical features and regarded them as manifestations of a disorder of

> language as an evolutionary human function associated with the development of a hierarchy of complex integrations in the nervous system and culminating in unilateral control by one of the two brain hemispheres (cerebral dominance).

Retardation in acquiring reading suggested that there was some interference with this natural process of growth and development.[104]

Orton and his disciples stimulated interest in children who were slow to read. Many papers appeared in the United States and in European countries describing patients who were "dyslexic," "alexic," or "retarded readers" suffering from "strephosymbolia." Many of these papers[20,88] reported children with symptom complexes very similar to those described by Orton.

Creak[20] described 38 boys and 12 girls with intelligence quotients upwards of 70 who attended a child psychiatric clinic. They were 2 years retarded in reading, compared with their mental ages as judged by standard tests. She noted that, compared with children in the normal population, a high proportion of patients showed mixed dominance, i.e., were left-handed and right-eyed or right-handed and left-eyed. She asked children to match shapes and patterns and reproduce them from memory and with the pattern in front of them. She tested their ability to listen to a simple rhythm and to reproduce it; many of her patients failed to do it. She found that reversals of numbers in reading and writing to dictation occurred less frequently than she had expected on the basis of the findings of Orton[103] and Monroe.[99] She noted the frequency with which psychiatric symptoms were found in children suffering from reading disabilities and speculated about the extent to which these symptoms were the result of reading difficulties and consequent unhealthy attitudes on the parts of parents and teachers and about the extent to which they contributed to educational failure. Unfortunately, a rather high proportion of her patients were of below average intelligence.

Eustis[42] described a family in which four generations showed an excess of ambidexterity, left-handedness, body clumsiness, retarded speech development, or other speech disorders and reading disability. He noted that "these conditions occur together in the individual and in his family tree sufficiently often to constitute a syndrome," but that all the symptoms he had described were found normally in infants and young children, and were to be regarded as abnormal only when they persisted into later childhood and adult life. Eustis[41] described 23 children with reading disability in 21 families. He found a family history of speech or reading difficulty in a high proportion of patients. The massive work of Hallgren[55] in Stockholm contributed significantly to the understanding of the genetics of "specific dyslexia." Hallgren found that 88% of the children he studied had a family history of speech or reading disorders.

In a series of studies, Hermann and his colleagues[59-61] reported on the deficient penmanship and abnormal spelling of children with "specific dyslexia." In writing, there was malalignment of letters, omission or repetition of words and letters, rotation of letters, odd punctuation marks, and misspellings. Typical faults were the partial or complete reversal of groups of letters and too short or too long linkages between letters. Sometimes letters ran into each other and children were especially likely to omit letters that were not sounded. He noted the marked tendency for children to write "phonetically" and related it to their poor memory for the shapes of words. He suggested that that might be attributed to some "underlying idiomotor apraxia." Hermann[59-61] considered that children with specific dyslexia could be defined as those having

> a defective capacity for acquiring at the normal time a proficiency in reading and writing corresponding to average performance; the deficiency is dependent upon constitutional factors (heredity), is often accompanied by difficulties with other symbols (numbers, musical notation, etc.), it exists in the absence of intellectual defect or defects of the sense organs which might retard the normal accomplishment of these skills, and in the absence of past or present appreciable inhibitory influences in the internal and external environments.

Hermann was struck by the similarity of the findings in children with "word-blindness" and those in Gerstmann's syndrome, in which right-left confusion, finger agnosia, dyscalculia, and agraphia were found. He wrote[58]:

> After having had the opportunity of examining a number of patients with Gerstmann's syndrome and having perused more than 1,000 case histories at the Ordblindeinstituttet, Copenhagen, I have no doubt that the fundamental disturbance in congenital word-blindness is the same as that of Gerstmann's syndrome.

Further case reports were made by Rabinovitch and co-workers[112,113]; Bender[6] and de Hirsch[28] noted that the "verbal visual configurations" of children suffering from specific dyslexia tended to remain unstable; "perceptual, visuo-motor and motor patterning often remains inferior. Their auditory discrimination is often poor, their Bender Gestalt is relatively primitive."

Relationship with Speech Development

Ingram and Reid[75] attempted to distinguish healthy, intelligent children from good environments with developmental dyslexia according to their performance on the Wechsler Verbal and Performance Scales. It was found that children whose scores on the Performance Scales were significantly inferior to those on the Verbal Scale more often showed errors of visuospatial type when reading and spelling, whereas those whose scores on the Verbal Scale were inferior more often had difficulties of audiophonic type. Some patients with a history of retarded speech development and poor performance on the Wechsler Verbal Scale had both visuospatial and audiophonic difficulties and were thus severely educationally handicapped. As in other similar surveys, a high proportion of patients had a positive family history of retarded speech development, stammer, and educational difficulties. Only 29% of patients were considered to have strong lateralization of handedness, and 52% were left-eyed. It will be seen that many of the findings in this study are similar to those reported by Creak.[20]

Some further evidence that patients with retarded speech development may have associated defects of visuospatial perception is found in the work of Doehring.[32] Doehring studied 20 children with severely retarded speech development who were diagnosed as "aphasic" and compared their ability to memorize the localization of a visual stimulus. He found that the accuracy of the performance of the aphasic group was significantly less than that of a matched group of deaf children or that of a group of normal children. He concluded that "children classified as aphasic are retarded in some, but not all, aspects of visual perceptual ability."

Kinsbourne and Warrington[79,81,83] took this work further. They developed a test of finger differentiation and demonstrated that 95% of children over the age of 7 years succeeded in identifying their fingers in the tests that had been developed. They postulated that children who showed relative inferiority of scores on the Wechsler Performance Scales, compared with the Verbal Scale, might show difficulties in finger differentiation more frequently than those with relatively inferior performance on the Verbal Scale. In particular, they thought that there might be an association between inability to recognize one's fingers and inability to succeed in subtests of the Wechsler Intelligence Scale for Children (WISC) with a high spatial loading.[92] They concluded that the differentiation of patients according to Ingram and Reid's criteria was valid and wrote[80]:

> It appears that among backward readers and writers there exist two groups with developmental defects reminiscent of acquired cerebral syndromes in adults. Group 1 will be called the language retardation group and Group 2 the Gerstmann group.
>
> The two groups are readily distinguishable by use of the finger tests. The Gerstmann group is further characterized by a positive discrepancy score on the WISC greater than that occurring naturally in 95 per cent of the population.

They considered that in Group 1 there were difficulties in the sphere of language, whereas in Group 2 poor progress in learning to read and write was attributable to difficulties in sequential ordering.

In further studies of the nature of the difficulties in the two groups of patients they had defined, they noted that children with retarded language development had a high proportion of extraneous letters in their writing to dictation, whereas patients with difficulties in finger differentiation produced a high proportion of errors related to letter order. They concluded that patients with a history of retarded speech development misspelled words in different ways and for different reasons than did those who had higher scores on the Wechsler Verbal Scale than on the Wechsler Performance Scales and associated difficulties in differentiating fingers. They considered that

> the association between finger agnosia and order errors in spelling appears to reflect an underlying, more general difficulty in processing information, both verbal and nonverbal, in terms of spatiotemporal sequence.[83]

Kinsbourne and Warrington,[83] like Ingram and Reid,[75] emphasized that there appeared to be a number of different clinical syndromes that resulted in reading and spelling backwardness of greater or lesser specificity.

Lateralization

Zangwill[137-139] discussed the significance of weak lateralization of handedness in dyslexia. He was skeptical of studies that merely compared the incidence of atypical laterality in matched groups of backward and normal readers, because he considered that reading retardation could have many different causes. He felt that it would be useful to ask whether there were particular types of reading backwardness associated with anomalies of laterality. He thought it likely that such differences would be found, and he wrote[139]:

> I have been struck by the frequency of retarded speech development, defects of spatial perception, motor clumsiness, and related indications of defective maturation in cases of dyslexia presenting in ill-lateralised (and some left handed) children.[138] In dyslexia presenting in fully right handed children (without familial sinistrality), on the other hand, I have been more impressed by the comparative "purity" of the disorder. It is in these latter cases, perhaps, that a specific genetical factor, as adduced by Hallgren[55] and Hermann,[59] might plausibly be assumed.

He considered that "minimal brain injury at birth" and "familial dyslexia" need not be mutually exclusive and that individuals lacking strong and consistent lateral preference might be particularly vulnerable to the effects of stress—"for instance, minimal brain injury at birth may affect more severely those who show no strong tendency to lateral specialization."[139]

Most authors report that significantly more boys than girls suffer from serious difficulties in learning to read and write. The male : female ratio is between 3 : 1 and 5 : 1 in most large series.[21] But in almost every other respect, there are major inconsistencies in the findings of authors who describe different series of patients suffering from specific dyslexia.

These are largely attributable to the different ages at which patients were studied and the very different means of selection. For example, Hallgren's patients came largely from remedial classes for children with retarded reading ability.[55] Patients described by Betts,[8] Eames,[37-39] and Park[107] were reported to have a high incidence of ophthalmologic abnormalities, but this may not be surprising in view of the fact that the authors were particularly interested in disorders of vision and visual perception. Most studies have shown that minor defects of acuity, visual fusion, and strabismus are not major causes of the types of reading disabilities that have been described. Abnormalities of eye movement during reading are almost certainly secondary to an underlying difficulty in visuospatial orientation, rather than primary causes.[21,55] A high incidence of emotional disturbances among patients referred to departments of child psychiatry is inevitable.[20,75,111] Drew,[35] a neurologist, found minor neurologic abnormalities in a father and his two sons who suffered from "familial congenital word-blindness," and suggested that minor neurologic abnormalities might have been found more commonly in more patients if they had been looked for—especially, perhaps, in series described by pediatricians.

NEW STUDIES

In a recent retrospective study, 176 children aged 7 or over were referred to the Department of Child Life and Health, Edinburgh, on account of suspected "dyslexia." They were referred from family doctors, school medical officers, and psychologists, and the majority had been examined in the Neurological and Speech Clinics of the Royal Hospital for Sick Children, Edinburgh; thus, they were highly preselected. Fifty-four were excluded from further consideration because they were found not to be failing in reading, and the apparent educational difficulties of another 19 were considered to be explicable on grounds of their being of below average intelligence or because of interrupted schooling or other environmental factors. Inadequate motivation and sensory defects appeared to explain the backwardness of nine children; eight failed to complete the testing and were excluded from the series; and four were too old for reliable diagnosis. There remained 82 patients who were considered to show reading failure as assessed by their scores on the Schonell Graded Word Test and the Stanford-Binet

Intelligence Scale 1960 Revision (Form L-M). Patients were considered to be mildly affected if there was a discrepancy of 10–14 points between their reading quotients and their intelligence quotients, moderately affected if there was a discrepancy of 15–29 points, and severely affected if there was a discrepancy of 30 or more points.

As a group, these children were reading at an average of more than 2 years below their chronologic age. Only three children were reading at the level of their chronologic age, but they were more than 3 years retarded in reading ability, commensurate with their mental age.

An attempt was made to classify children by the likelihood of their suffering from "brain damage" according to their histories and indications of brain dysfunction ascertained in the course of detailed neurologic examination and electroencephalography. Birth histories were judged arbitrarily in the same way as were those of patients suffering from cerebral palsy in a previous study.[71] They were scored for the possible and probable traumatic, hypoxic, and toxic insults that they might have suffered *in utero.* Thus, mild or moderately severe pre-eclampsia was scored as one hypoxic insult, severe pre-eclampsia or antepartum hemorrhage or prolapse of the cord as two hypoxic insults. Midcavity forceps delivery was counted as one traumatic insult, but labor prolonged beyond 36 hr in a first gestation or beyond 24 hr in a second gestation, breech extraction, and high forceps delivery were counted as two traumatic insults each. Apnea up to 5 min was considered as one hypoxic insult, more prolonged apnea or cyanosis for more than an hour as two hypoxic insults. Patients who were born after uncomplicated pregnancies, labors, and deliveries and who were normal in the neonatal period were placed in category 0, those with one or two insults in category 1, those with three or four insults in category 2, and those with five or more insults in category 3.

A history of "clumsiness" or delayed motor milestones sufficiently severe to make the parents seek medical advice placed a patient in category 1. A history of convulsions with fever, strabismus, or other conditions frequently associated with chronic brain disease placed a patient in category 2. A history of definite head injury, meningitis, or encephalitis or of persistent neurologic impairment, such as ataxia or neglect of a hand, placed a patient in category 3.

Patients were also classified according to the findings of neurologic examination. Patients without abnormal neurologic signs were placed in category 0. Those with minimal incoordination or a mild excess of contralateral associated movement, for example on the Fog test, were placed in category 1; they probably had no more than a degree of neurologic immaturity.[51] In category 2 were those with more evidence of motor impairment, particularly those showing the choreoid syndrome or mild asymmetries in the reflexes without definite paresis. Patients with definite cerebral palsy or with epilepsy were placed in category 3.

The electroencephalograms of patients were also classified into four categories. Normal records for age were in category 0; records that showed a mild excess of slow activity or minor asymmetries but no paroxysmal activity were in category 1; records in which there was a definite excess of slow activity that might be paroxysmal were in category 2; and records with focal spike activity, spike wave activity, or other evidence of epilepsy were in category 3.

In assessing the likelihood of underlying brain damage, most weight was given to the results of neurologic examination, for that seemed to be the most informative method of study and did not rely on retrospective findings.[74]

As stated, all patients were given the Stanford-Binet Intelligence Scale 1960 Revision (Form L-M). All but six, for whom reports from the school authorities were accepted, were given standardized tests in mechanical arithmetic (Schonell Form A or B), and the Schonell Spelling Test (S 1) was administered. The Goodenough Draw A Man Test was given to 49 children under the age of 10 years. Children were placed in one of two categories according to the results of these tests. In the first category were 62 children who appeared to have specific reading and spelling difficulty only (referred to for the sake of brevity as "specifics"). In the second were 20 children who failed in reading, spelling, and arithmetic (referred to as "generals" because they had general learning difficulty in spite of favorable mental, emotional, and environmental factors).

The "specifics" and "generals" were similar in age, social class, and average IQ. The composition of the subgroups differed appreciably only in the sex ratio, the "generals" showing a higher proportion of girls than the "specifics." More than 50% of patients gave a history of slow speech development, the percentage being similar in the two groups. About 40% of the "specifics" and 25% of the "generals" had a family history of reading and spelling difficulties in sibs, parents, or aunts and uncles; 66% of those with a positive family history of reading difficulties were in the category of severe reading failures. All the "generals" with positive family histories were boys, and all were severe reading failures. In a slightly comparable group of 30 children of equivalent intelligence and social class (but not comparable in other respects), who read normally, a family history of reading difficulty was found in only 9%. The "specifics" had less evidence of brain damage or dysfunction than the "generals," whether this was assessed on the basis of birth and developmental history, clinical examination, or electroencephalography. Thus, 68% of the "specifics" and only 35% of the "generals" were considered to have no significant neurologic abnormalities; 22% of the "specifics" and 60% of the "generals" had significant neurologic abnormalities. About 66% of the "specifics" had normal electroencephalograms, compared with only 16% of the "generals." These are differences of statistical significance.

An attempt was made to classify the types of reading error made by patients in the two groups. Difficulties in learning to recognize written symbols, associate them with the corresponding auditory image, and then synthesize them into words are characteristic of the early stage of reading. At a later stage, visual recognition of words becomes immediate, and finally the child reads by making a series of hypotheses, often on slight contextual clues, and learns to "scan" a line of print or script.[87,134]

All the "specifics" made errors characteristic of the early stage of reading, involving confusion of letters or audiophonic synthesis. These were found less frequently in the "generals." Although 77% of all "specifics" made audiophonic mistakes, only 10% of the "generals" did. In 11% of the "specifics" only audiophonic mistakes were made, whereas none of the "generals" made only that primitive type of error. Audiophonic difficulties of this type were very striking in patients in the "specific" group who had a history of retarded speech development, but they were not the only cause of their reading failure. For example, one 8-year-old in the "specific" group who had undergone entirely normal development, had normal neurologic findings, and was of very superior intelligence could not recognize the simplest words in print—such as "dog," "man," or "my." In such cases, some inadequacy of visual recognition must be postulated.

Although evidence of brain damage was usually, but not exclusively, associated with dyslexia and additional educational difficulties, it was not associated with a greater degree of difficulty in learning to read: 70% of the "generals" who had no evidence of brain abnormalities showed severe reading failure, but only 57% of those with definite evidence of brain abnormality showed severe reading failure. Furthermore, among the "specifics," 60% of those with normal neurologic findings had severe reading failure, compared with only 33% of those with definite evidence of brain disease.

It is impossible to place the patients described in clearly defined categories on the basis of the findings reported. It is evident, however, that specific reading handicaps may be present in association with no clinical or electroencephalographic indications of brain abnormality, with definite clinical indications of brain abnormality, or with neurologic signs that are more suggestive of neurologic immaturity than of serious focal disease.

When there are definite clinical signs of brain abnormality, general educational difficulties are more likely to be found than isolated reading retardation, and the family history is less often positive for reading and spelling disorders.

Even in the group of patients with specific reading disability without clinical evidence of brain disease and with a positive family history of reading and writing difficulties, it is possible to discern different groups—for example, those whose errors in reading and spelling are predominantly visuospatial and those whose errors are predominantly of the audiophonic type, with the latter more often having a history of retarded speech development.[74] This survey, being retrospective and dealing with a highly selected population, is not very different from a number of others that have been described previously in this paper, but the findings do indicate that "specific developmental dyslexia" should not be regarded as a single disease entity. Moreover, it suggests, in agreement with Zangwill, that "hereditary dyslexia" and "dyslexia due to brain injury" are not necessarily mutually exclusive categories.[139]

It is apparent that there is a need for detailed studies of carefully selected groups of children with reading difficulties from the neurologic, ophthalmologic, psychologic, and psychiatric points of view, so that the various syndromes associated with more or less "specific dyslexia" or "specific developmental dyslexia" may be further defined.[98] At the same time, it is only through comprehensive surveys of representative groups of schoolchildren that the relative importance of these syndromes in causing retardation can be assessed.

POPULATION SURVEYS

While clinicians were publishing large numbers of case reports about children considered to suffer from specific dyslexia, psychologists and educationalists were attempting to estimate the size and nature of the problem of reading backwardness in large school populations.[16,99,120,121,129] Monroe,[99] reporting as a former research associate of Orton, found 15 possible "causative factors" in reading disability. She recognized two major types of disability: "a lack of precision in discrimination of complex visual patterns," shown in the child's reading by his inability to comprehend words as units, although he might be able to recognize them when they were spelled out to him; and "a lack of precision in discrimination of spatial orientation," similar to the tendency to reverse symbols and the order of written symbols described by Orton[103] as "strephosymbolia."

MacMeeken[88] studied 383 children in a large Edinburgh primary school between the ages of 7 years 6 months and 10 years 5 months. In her words, "a cross section would show that the children come from homes fairly far down but not very far up the social scale." MacMeeken gave individual tests of intelligence and reading ability. She obtained a reading-accomplishment quotient by using the formula, (reading age/mental age) × 100, and considered that those with quotients of less than 85 were suffering from reading disability and those with quotients of between 85 and 90 were "handicapped." Of the children she tested, 9.1% had quotients of 85 or less: 12.2% of the boys and 6.2% of the girls, a difference that was almost statistically significant. The mean intelligence quotients of children with reading disability were 106.3 for boys (range, 83–137) and 101.9 for girls (range, 86–121). A very high proportion showed left-eye dominance, although the distribution by handedness was not different from that of children in the general population. MacMeeken recognized that "absences, changes of school, defective eye sight, defective hearing may and do produce reading achievement which is low in relation to mental capacity," but she considered these causes insufficient in themselves to produce disability of the severe degree that she found, although they might be contributory causes. She considered that

> these children are developmental aphasics showing in varying degree and as major aspects of their pattern of difficulty the two aphasic syndromes of word recognition

difficulty and the strephosymbolic tendency to reverse letters, to distort letters or syllables within words.

Her findings may be compared with those obtained in a survey of primary-school children in Stockholm carried out by Hallgren,[55] whose conclusions were broadly similar, although he found a lower incidence of "left-eye dominance."

In a study of children in England, the Ministry of Education[97] found that 1% of 18-year-olds, 1.4% of 15-year-olds, and 4.2% of 11-year-olds had reading ages of less than 7 years. Some 3 years later, a survey[96] was carried out in Middlesborough after programs of remedial reading had been established. A somewhat smaller proportion of the population of 11-year-olds had reading ages of less than 7 years: 3.9% of boys and 1.3% of girls. In Leeds, 19% of 1985 11- and 12-year-olds, excluding those who attended grammar (i.e., public) schools, were more than 2 years retarded in reading, as judged by chronologic age, and 8.6% were 2 years retarded, as judged by mental age.[19]

More recently, Morris[101] noted that 19.2% of 7-year-olds in Kent County, England, were still using their first reader, which implied that they had hardly begun to learn to read.

A nationwide study[77] of 11,000 British 7-year-olds found that 11.2% of boys and 5.9% of girls had not progressed beyond their first reading book after 2 years of elementary-school instruction. The difference in the proportions of boys and girls with reading retardation was statistically significant.

In the United States, Eisenberg[40] studied the reading proficiency of the entire sixth-grade population (aged about 12 years) in a large urban center. Some 28% were found to be two or more grades below their expected level, whereas in "commuter county," 15% were two or more grades below expected level, and in superior independent schools, 0% (Table 1). In the independent schools, 82% were two or more grades in advance of their expected reading ability. Eisenberg, like the authors of the other population studies quoted, emphasized the multiplicity of factors that could produce reading disability—sociopsychologic: quantitative and qualitative defects in teaching, deficiencies in cognitive stimulation, and deficiencies in motivation (associated with social pathology or with psychopathology); and psychophysiologic: general debility, sensory defects, intellectual defects, brain injury, and specific (idiopathic) reading disability.

TABLE 1 Reading Levels of Sixth-Grade Children by School System[a]

School System	Test	% Retarded ⩾2 Grades	⩾1 Grade	% Advanced ⩾2 Grades
Metropolis	Stanford	28	57	9
Commuter county	California	15	35	8
Suburbia	Iowa	3	19	34
Independent	Stanford	0	1	82

[a] Derived from Eisenberg.[40]

British workers have similarly been reluctant to subscribe to the view that specific developmental dyslexia is a major cause of reading disability. Burt[16] refuted the idea that specific dyslexia was of major importance, and Vernon[127] wrote that "investigations which have been cited give no clear evidence as to the existence of any innate organic condition which causes reading disability except perhaps in a minority of cases; though certainly innate factors may predispose the child toward difficulty in learning to read." She appears to have regarded specific dyslexia as an irremediable condition, and many of the studies she quotes describe patients who probably would have been diagnosed by many clinicians and psychologists as suffering from "specific dyslexia." Nevertheless, she admits that "there may be a class of individuals who are generally lacking in maturation, these are the cases . . . who have no well established laterality and in addition exhibit speech and other motor disorders, temperamental instability and reading disability." Later, she modified her point of view somewhat and appeared to be prepared to admit that "specific dyslexia" may be more important as a cause of reading disability than she had originally thought.[128]

Daniels[25] laid stress on the fact that virtually all children with severe reading retardation could be helped by appropriate remedial teaching and used this as an argument against the existence of any such condition as "congenital word blindness." This argument, however, is not generally accepted as valid by psychologists and remedial teachers who have experience with large numbers of so-called "dyslexic children," most of whom are found to make good progress with appropriate treatment.[10,26,27,31,43,44,116]

It is difficult not to feel, with Critchley,[21] that the picture that has developed in the minds of many educationalists and psychologists–that specific dyslexia is an irremediable, clearly defined disease entity with constant symptomatology and signs–is responsible for their not recognizing the prevalence of the syndromes of specific dyslexia described largely by medical authors.[21,22] To some extent, however, medical authors have contributed to this misconception by overemphasizing the constancy with which, for example, directional confusion, mixed laterality, and a positive family history of reading disorders are found.

In a recent, more open-minded study of the total population of 9- and 10-year-olds in the Isle of Wight, Rutter *et al.*[119] found that 4% of children had reading ages measured on Neale's tests that were at least 28 months below their mental ages as assessed by the WISC test. The mean Wechsler IQ of the children was 103.[118,136]

> It was found that specific reading retardation was associated with considerable difficulties in arithmetic, severe problems in spelling, delay in the development of language and immaturity of language still evident on examination to age nine and ten years; inco-ordination; motor impersistence; right/left confusion; (possibly) difficulties in constructional tasks; and a strong family history of reading difficulties. There was no association between left handedness or mixed handedness and specific reading retardation. With this one exception, however, all the features said to be characteristic of "dyslexia" were found to be associated with specific retardation.[119]

Rutter[118] studied the clustering of "dyslexic characteristics in individual children showing speech and language abnormalities, motor incoordination, constructional difficulties, motor impersistence and right/left confusion." None of the 86 children with specific reading retardation showed all five characteristics, only five showed four of them, nine showed three, 20 showed two, 29 showed just one, and 23 children showed none. Rutter concluded that there was no general tendency for the items to cluster together and that the associations between the various developmental abnormalities were of a low order. He felt that his findings offered no support for a single syndrome of "dyslexia." A positive family history of reading difficulties was not found more frequently in those with specific reading disability. Rutter recognized that these findings did not exclude the possibility that various syndromes were associated with difficulty in learning to read and considered that, "whatever the basic cause of the different types of specific reading retardation, the final state is frequently the result of a combination of biological, social and psychological factors." His ideas conform with those advanced by workers who believe that a number of syndromes with characteristic neurologic and psychologic findings may contribute to the difficulties in learning to read found in children in any large population.[40,70,75,109]

STUDIES OF CHILDREN SUFFERING FROM BRAIN DAMAGE

Studies of children suffering from cerebral palsy and other syndromes associated with nonprogressive brain damage sustained before, during, or shortly after birth inevitably influenced contemporary thought about the nature of reading and spelling difficulties in childhood. Lord[85,86] emphasized the lack of correlation between mental age and educability in patients suffering from cerebral palsy and described the perceptual and visuomotor difficulties from which many children suffered, including some with relatively high intelligence. She observed that many dyskinetic children (in whom involuntary movements were the presenting feature) could draw, whereas other spastic children, whose motor functions were less impaired, could draw less well because of associated perceptual difficulties. Strauss and Lehtinen[123] summarized Strauss and Werner's work[124] with figure background tests (marble board, tachistoscopically exposed pictures, tactual motor tests) and other tests in children with brain damage, in many of whom behavioral abnormalities and epilepsy were shown, but not cerebral palsy. Cruickshank and Bice[23] attempted to correlate the nature of the failures on psychologic tests with the different clinical diagnoses of cerebral palsy and the clinical severity of the condition.

A more extensive review of the perceptual difficulties of children suffering from cerebral palsy, particularly figure–background relationships, was made by Cruickshank *et al.*[24] They studied 110 nonhandicapped children and 325 children between 6 and 16 years old who suffered from cerebral palsy and had a minimal mental age of 6 years and IQ within average for age. Six tests of tactile and visual perception were given to the children. It was found that cerebral-palsied children made consistently poorer performances than the nonhandicapped children in four of the six tests administered. Spastics had more difficulty in carrying out the tests than children suffering from choreo-athetosis (dyskinetic cerebral palsy). The relationships between the tests were low, but performance on the tests did correlate with chronologic age.

Floyer[50] studied 72 cerebral-palsied children who were considered educable. The group was matched with normal children for sex, IQ, and chronologic age. It was found that tests most sensitive to lag or disturbance in visuospatial perception tended to be poorly performed by the patients who suffered from cerebral palsy, especially the girls. They had difficulty in visuomotor tasks, such as assembling pieces of form board, block building from memory, copying simple designs on paper, and doing the progressive matrices of Raven.[114] Floyer, like many other authors, emphasized that many of the difficulties on psychologic testing that she found in patients suffering from cerebral palsy could be interpreted in terms of immaturity of functioning, rather than permanent disturbance of function that could be attributed to a particular brain lesion.[2,33,34,125]

The question as to how far the visuospatial difficulties and the associated difficulties in reading and spelling may be regarded as manifestations of developmental lag in concept formation or as specific deficits unlikely to disappear with maturation is fully discussed by Abercrombie,[1] who concluded that

> it would seem that as far as perception is concerned, there is little evidence that cerebral palsied children see things in a distorted manner, though they may see them in a primitive or immature way, that is, they fail to make differentiations at the level of complexity which might be expected from their mental age. They can therefore be described as suffering from developmental lag in their perceptual skills. The bizarre drawings and other constructions which some of them make may seem to be unlike what normal children produce at any age, but it is possible that these also may be explained by a mixture of immature and more mature ways of performing.

She notes, however, that although there is some evidence that the developing brain is functionally disordered by localized damage in less specific ways than the adult brain, it is perfectly possible that different kinds of perceptual constructive or cognitive difficulties resulting from chronic lesions at different sites might be revealed by more sophisticated psychologic tests. Some evidence for the latter view may be found in the work of Wedell.[132,133]

Birch and Lefford[9] and Bortner and Birch[11] emphasize the way in which tactile, visual, and auditory perceptions modify each other and affect behavior as children mature. They write[9]:

> The evidence for normal children strongly confirms the view that the elaboration of intersensory relations represents a set of developmental functions showing age-

> specific characteristics and markedly regular curves of growth. At the very least, the emergence of such relationships appears to be delayed in the "brain-damaged" children, a factor which may seriously limit possibilities for the normal utilization and integration of environmental information.

They found that a high proportion of their "brain-damaged subjects" showed severe impairment of intersensory perception, which did not appear to improve between the ages of 5 and 18 years.

Prechtl and Stemmer[110] attempted to pick out a uniform neurologic syndrome among the many heterogeneous groups of hyperkinetic children who had been referred to a neurologist because of poor school performance. Fifty children whose major abnormality on neurologic and electroencephalographic examination was found to be the presence of choreiform movements were studied in detail. A high proportion showed additional minor neurologic abnormalities on careful examination. Of the 50 children, 90% had "more or less trouble in reading." They were frequently described by their parents and teachers as being abnormally clumsy, distractible, and restless; they found sitting still or concentrating on a subject for long periods extremely difficult. Prechtl and Stemmer were inclined to attribute the learning difficulties of this group of children suffering from the "choreiform syndrome" to difficulties of visual fixation, instability of concentration, and a lag in the development of cerebral dominance and "complex functions." A high proportion of such patients score very poorly on tests of visuospatial perception and, as noted by Birch and Lefford,[9] have difficulties in intersensory integration. A positive family history of difficulties in learning to read and spell was not noted in the account given by Prechtl and Stemmer of their studies.[110]

It is clear from the investigations of visuospatial and intersensory perception in brain-damaged children described above that a variety of handicaps that impair children's ability to learn to read and spell are found frequently. The extent to which their perceptual defects can be attributed to maturational lag and to damage to particular areas of the developing brain is still a subject for much discussion.[80,82,87] Certainly, it is difficult to distinguish the reading and spelling errors made by children with known brain damage from those made by children with no evidence of brain damage but with a positive family history of reading and spelling difficulties.

The finding that a high proportion of children who suffer from cerebral palsy and other manifestations of brain damage are slow to speak, have difficulties in learning to read and spell, and may perform poorly on tests of auditory and visuospatial perception has given rise to much speculation as to the causes of reading and spelling difficulties in the whole child community.[69, 72, 73]

PROSPECTIVE SURVEYS

Retrospective studies of series of children suffering from reading disability have many drawbacks. If they deal with large population samples, individual pupils tend not to be fully investigated by all the methods available in psychologic and medical research. Smaller populations are more manageable, but children with significant difficulties in learning to read and spell are few and tend to be very heterogeneous, so that firm conclusions about the causes and nature of their difficulties cannot be made. Prospective studies in which groups of preschool children are selected for investigation by clinical and psychologic methods before they go to school and then re-examined and retested in their early school years are of great value. Three studies of this type will be described.

Completed Studies

The first study is described in *Predicting Reading Failure* by de Hirsch *et al.*,[29] which gives an account of a study of 53 children who came from homes in which English was the predominant spoken language, had IQ's within one standard deviation of 100 as obtained on Form L of the Stanford-Binet Intelligence Scale (1937 Revision), and showed no significant sensory defects and no evidence of psychopathology. A wide variety of tests of "behavioural patterning, motility patterning, gross motor patterning, fine motor patterning, laterality, body image perception, visual perception patterning, auditory perceptual patterning, receptive language tests, expressive-language tests and sentence development" were given, in addition to reading-readiness tests, while the children were in kindergarten. There were 37 tests in all. After 1 year at school, all the children were given standardized tests of reading, spelling, and writing; after another year at school, tests were repeated.

The aim of de Hirsch and her colleagues was to develop a battery of tests that could be given in the kindergarten period and could predict which children were likely to suffer from reading and spelling difficulties in later years. They found that a series of 10 tests–ability to use a pencil, the Bender Visuo-Motor Gestalt Test, the Wepman Auditory Discrimination Test, the Number of Words Used in a Story Test, tests of categorization, the Horst Reversals Test,[67] the Gates Word Matching Test, two tests of word recognition, and one test of word reproduction–gave scores in the kindergarten period that correlated closely with the scores obtained on standardized tests of reading and spelling given at the end of the first two grades of schooling (ages 7 and 8). Eight children were placed in the class of "failing readers." They comprised the lowest 15% among the children tested; all of them scored zero on the Gray Oral Reading Test at the end of the first grade, and five of them scored zero again a year later. Six of the eight failing children were boys; five were noted to be "markedly hyperactive, distractable, impulsive and disinhibited; they needed many opportunities to move around the room, and became resentful when they were required to sit still." Interestingly, they showed no more ambilateral responses than did the other 45 children, but "the auditory-perceptual and oral-language tools of the failing readers were decidedly inferior to those of the remaining subjects." Four boys and four girls were categorized as "slow starters." They scored zero on the Gray Oral Reading Test at the end of the first grade, but achieved their expected level at the end of the second grade. Their reading and spelling difficulties appeared to be transient; as they developed and received instruction, their performance improved. Two boys and six girls were considered to be "superior achievers" both in reading and in spelling.

De Hirsch *et al.* commented that

> the failing reader's perceptual, motor and linguistic responses were strikingly unstable. As do chronologically younger children they functioned at a primitive and an undifferentiated level. Their fragmented figure drawings, their poorly synthesised Bender designs, their inability to organise parts of a story into a meaningful whole suggested a relatively low level of integrative competence.
>
> It was not, however, any failure on any single task that distinguished the failing readers from the other subjects but rather the accumulation of deficits.

They thought the "accumulation of deficits" pointed to severe maturational lags. They argued that the relatively poor scores of prematurely born children, compared with those of children born at term and having equivalent intelligence, reported by Kawi and Pasamanick[76] and Drillien[36] and confirmed by their own studies, might also be explained on the basis of retarded maturation of higher nervous activity:

> In summary these findings as well as clinical experience support our supposition that there is indeed a close link between a child's maturational status at kindergarten age and his reading and spelling achievements several years later.

A relatively small but very detailed longitudinal study of the reading and spelling handicaps of 56 boys attending an upper-class school in the United States has recently been presented by Rawson.[115] Rawson rated the severity of difficulties in learning to read and write in her pupils and considered that there was a continuum of language ability between those who were superior and those who could be considered "dyslexic." She categorized 20 children as having high, 16 medium, and 20 low language facility. Of the 20 children with low language facility, eight were categorized as suffering from moderate dyslexia and 12 as suffering from severe dyslexia. It is to be noted, however, that four of the 12 in the latter category were admitted to school after the first grade because there was a language re-education program in the school that the children's parents wanted to take advantage of.

Rawson followed her pupils into adult life and was able to show that even those who seemed most handicapped by language disability achieved university degrees and professional positions in society in later life at least equal to those of their school fellows with high language-learning ability, even though they might have some residual difficulties in reading rapidly and especially in spelling conventionally. It is interesting to note how aware of their minor persisting difficulties many of her ex-pupils were as adults. Like MacMeeken,[88] Rawson felt that difficulties in the environment contributed to, rather than caused, reading and writing difficulties.

New Findings

In 1961, the Medical Research Council supported a program of research into the problems of children 2½–5 years old with retarded speech development and their later educational problems in the Department of Child Life and Health, University of Edinburgh. As noted earlier, a number of papers had indicated that a high proportion of children with slow speech development later had difficulties in learning to read and write.[20,42,46,93–95,103] Rawson[115] reports speech development problems in nine of her 12 most severely dyslexic subjects. Of the 78 children considered to suffer from developmental dyslexia studied by Ingram and Reid,[75] more than half showed retardation of speech development; less formal studies of children in a speech clinic demonstrated that a high proportion of them had later difficulties in learning to read and write.[70]

To see whether it was possible to isolate one relatively "pure" type of dyslexia, it was decided to investigate what happened to a series of children with significantly retarded speech development once they went to school. The subjects were a group of 73 children in whom a diagnosis of speech retardation was made in the Speech Clinic of the Royal Hospital for Sick Children, Edinburgh, and subsequently confirmed on the basis of tests of articulatory development and language development in the Department of Child Life and Health, University of Edinburgh. So that the effects of environmental influences and, in particular, social differences would be minimized, children of only the two upper social classes (I and II according to the Registrar General's classification) were chosen. A control group of 140 children of similar class was obtained, so that variables that might adversely affect the learning of reading and spelling (other than speech retardation in the clinical group) might be excluded. Excluded from both groups were children with any history that gave rise to suspicion of brain damage, abnormal behavioral development, hearing impairment, behavior disorders, or any neurologic abnormalities on examination. The hypothesis was that healthy children of average or superior intelligence who showed retarded speech development would later have more difficulties in learning to read and spell than a comparable group of children without retarded speech development.

This study is still incomplete, because it is impossible to test reading and spelling performance accurately before children have been in school at least 2 years. Up to October 1968, 78 control and 51 speech-retarded children had been tested after being in school for at least 2 years. Some preliminary results are shown in Table 2.

Because the average IQ of patients in the control group was a little higher than that of the speech-retarded patients, 29 children in each group were matched for sex and Stanford-Binet IQ. These matched subgroups showed the same differences as the entire group.

There were very significant qualitative differences between the chil-

TABLE 2 Analysis of Preliminary Data (to April 1968) on Later School Attainments of Speech-Retarded Children[a]

Skill	Group	No. in Group	Backward[b]	
			No.	%
Reading	Speech-retarded	51	15	29
	Normal control	78	1	1
Spelling	Speech-retarded	51	18	35
	Normal control	78	5	6
Arithmetic	Speech-retarded	51	4	8
	Normal control	78	6	8

[a] Derived from Mason.[90]

[b] *Absolute backwardness* is considered to be indicated by educational age 6 months below chronologic age of 7 years irrespective of IQ.

dren who had a history of speech retardation and those with normal speech development. Only nine (18%) of the 51 speech-retarded children made a good start in reading; 14 (27%) started badly but seemed to "catch up" and had a reading quotient within 15 points of IQ at the time of test; the remaining patients were retarded in reading attainment. In comparison, only 15% of the control group of patients had difficulty in the initial stages of learning to read. These findings may be compared with those of de Hirsch.[27]

All the speech-retarded children except the nine (18%) who succeeded in reading from the start gave evidence of perceptual inadequacy[120] of either visuospatial or audiophonic type. Of the 16 children who failed completely in learning to read, three had severe visuospatial difficulties, two had severe audiophonic difficulties, and 11 had difficulties of both types. Of 14 children with mild, moderate, or severe failure, four had visuospatial difficulties, six audiophonic, and four both. Of 12 who had problems in the early stages but were reading satisfactorily at the age of 7, four had continuing audiophonic difficulties with or without visuospatial difficulties, and the other eight had lost the audiophonic disabilities that had previously troubled them. This finding supports Schonell's observations[120] that audiophonic difficulties diminish in severity and frequency with increasing age. It also indicates that audiophonic inadequacy for reading may arise from a developmental lag, rather than from chronic inadequacy.[56,135]

It was not surprising to find that the subjects who suffered from slow speech development, many of whom had residual speech defects, experienced audiophonic difficulties when they came to learn to read. It became increasingly apparent as the study proceeded, however, that many of the difficulties in learning to read suffered by the subjects with slow speech development were not of audiophonic type, but visuospatial. Mason[91] made the interesting observation that children with speech retardation, tested before they had reached school age, showed significantly depressed scores on the Goodenough Draw A Man Test, compared with the control group. Mason considered that the relative failure of the subjects in the speech-retarded group could be considered as one aspect of maturational lag, inasmuch as the subjects "drew a man" in the same way as children in the control groups would when 2 or 3 years younger. The extent to which their relative inability to draw a man could be attributed to visuospatial disabilities and the extent to which those disabilities could be related to deficiencies in conceptualization are subjects for speculation.[29,54,90]

The interim findings of this still incomplete study indicate that speech-retarded children in the upper social classes in Edinburgh, irrespective of their level of intelligence, are at a high risk of failing to learn to read and spell as quickly as children of similar intelligence and social class without a history of speech retardation. The risk of reading failure is as great as 75% when the degree of speech retardation is 18 months or more at the age of 4½ years. At least 16% of the speech-retarded children so far tested meet the definition of a child suffering from "specific dyslexia"–i.e., a child who, in the absence of impaired intelligence, physical defects, poor motivation, or adverse environmental factors, nevertheless experiences severe difficulty in the learning of reading although he performs satisfactorily in the early school years in subjects independent of reading. The proportion of children considered to suffer from "specific dyslexia" is likely to increase considerably as the study proceeds.

Reading disability did not appear to be associated with any evidence of organic disorders in the patients studied; rather stringent neurologic examination had failed to reveal any indications of brain abnormality. But the reading and spelling difficulties could not be attributed to environmental difficulties. There appeared to be evidence of constitutional reading disabilities. These disabilities, however, did not take a single form. Some children were retarded in reading because they were unable to perform adequately in the audiophonic tasks demanded by reading, others because they had visuospatial difficulties. In a third group, both types of difficulty were apparent. Preliminary data suggest that the outlook for overcoming or outgrowing reading and spelling disabilities is most favorable when the difficulties are audiophonic.

DISCUSSION

The Reading Process

What is known about the complex processes by which children learn to read and write with accurate spelling has been fully reviewed by Vernon,[127,129] Schonell,[120] Burt,[16,17] and, from rather different standpoints, Vygotskii,[130] Piaget,[108] and Luria.[87]

Luria[87] points out that the process of reading begins with the perception of letters and then the analysis of their conventional phonetic value. "This is followed by a very complex process, causing the most obvious difficulty in education—the process of fusion of the phonetic letters into words." Isolated phonetic sounds have to be recoded into complete syllables before they can be combined into whole words. As reading skill develops, the "analysis and synthesis of individual phonetic letters is gradually contracted and simplified and is eventually transformed into the direct recognition of words by sight."

Luria considers that this is a characteristic of fully developed reading skill, the third stage. This stage of reading skill is reached when the reader perceives immediately the likely meaning of groups of letters or words and begins to "scan" sentences. He learns to compare the expected meaning from minimal clues given in the text with the meaning actually expressed, and it is only occasionally with difficult words that he has to resort to the earlier methods of detailed phonetic analysis and synthesis. The process by which the child develops "guessing reading" or reading sentences or even paragraphs on the basis of minimal clues is crucial to the development of fluent reading characteristic of literate adults.

To recognize letters, some minimal visuospatial abilities are required.[52,53] Vernon[127] points out that, to identify words, a child must be able to recognize individual letters and perceive their ordering in space if he is to be in a position to sound them correctly. In most cases, while they are acquiring the ability to do that, most children learn to recognize some words by their characteristic shapes, and the "guessing reading" process described by Luria begins.

Vernon notes that small children may not realize what details of shape and form are essential, and they are likely to fail to understand relationships of parts of a word to the whole word. In particular, they have difficulty in perceiving the significance of the ordering of words. Defects of this type have been demonstrated in a number of studies on children who are retarded in learning to read and spell.[18,54] They tend to confirm the statement by Hermann[59] that in many cases there is a fundamental disturbance "involving the categorical sphere of function which may be termed directional function":

> The directional disturbance is related to a failure of lateral orientation with reference to the body-scheme, such that concepts of direction are either uncertain or abolished; the individual consequently has difficulty in orientating himself in extrapersonal space. This difficulty in orientation has particular consequences for the ability to operate with symbols such as letters, numbers, and notes.

A number of authors have commented on the similarity of the processes involved in distinguishing temporal sequences that determine the order of speech sounds in oral language and spatial sequences that determine the order of written symbols in written material.[89] Ilg and Ames[68] found that transposition of letters within words and complete word reversals persisted considerably later than letter reversals in reading and that was also true of children who had had difficulty in learning to read when they came to write. Other forms of substitution, however, were fairly frequent, especially within monosyllabic words.

At some stage in reading development, children learn to identify word shapes in association with related word shapes in the sentence; they begin to read from general clues rather than from item analysis. The ages and stages at which this process begins to occur are variable from child to child, but it seems to be relatively late in children who are slow to read.

The ways in which children learn to correlate visual symbols with sound symbols have been studied extensively, but no major conclusions have been reached. It is obvious, however, that, at some stage in their process of learning to read, children must learn how to correlate written symbols with their spoken equivalents. In English, as Vernon[127] has described, there are great difficulties in doing so because of the large number of unsounded written symbols and the variation in vowel sounds in different positions. Another difficulty is that sounded words make sense only in context. As Vernon writes:

> Thus the child has to learn to understand that (a) each word and its sound patterns are separate entities, with their peculiar, invariable and universal characteristics; (b) each word's sound pattern can be analysed into a succession of sounds with a characteristic and invariable sequence; (c) these unitary sounds can be generalised, in the sense that they occur in approximately the same form, but in different sequences, in different words; (d) the sounds correspond to different letter shapes visually perceived; but (e) unfortunately in the English language the relationships between sounds and visual percepts vary considerably from word to word.

Vernon is somewhat cautious in attributing difficulties in learning to read and spell to audiophonic disabilities but admits that "the one universal characteristic of non-readers suffering from specific reading disability is their complete failure to analyse word shapes and sounds systematically and associate them together correctly."

> Why even quite an intelligent child should fail to realise that there is a complete and invariable correspondence between printed letter shapes and phonetic units remains a mystery which, as we shall see . . . , has not been solved. It must be attributed to a failure in analysing, abstraction and generalisation but one which, typically, is confined to linguistics. Perhaps the obvious syncretism and familiarity of spoken words makes it particularly hard to apply cognitive reasoning to their analysis. It is certain that in some cases only a drastic form of analysis proves efficacious such as that utilised in the Fernald and Keller Tracing Method.[45]

Variety of Disturbances

It is apparent that the complex and interrelated functions required for the normal development of reading and spelling may be disturbed in a wide variety of ways. Children who have difficulties in recognizing shapes, patterns, and sequences of patterns are likely to have trouble when they are asked to identify letters in different sequences. Children with difficulties in recognizing sequencing, whether in spatial or temporal terms, are likely to have difficulties in conceptualizing sequences of written symbols or sound symbols and in transmitting one to the other.[89] Difficulties in sequencing are likely to be more evident when the child tries to spell than when he tries to read.

Difficulties in correlating the written with the spoken symbol are almost invariably found in children with reading and spelling difficulties. It is difficult to account for them in modern theories of learning, but the idea that sequencing in either temporal or spatial relationships may be impaired does make it possible to understand to some extent how both visuospatial and audiophonic difficulties may arise. Difficulties in relating written symbols to their spoken equivalents are found very frequently in children with slow speech development and in most of them are the major cause of retarded development of learning to read and write. In 1957, it was possible for Vernon[127] to write:

> Speech defects and slow language development have often been found in backward readers and may have been contributory to the retardation, but in the experimental study of these defects there has been no attempt to determine the frequency with which they appear in severe cases of disability as distinct from their occurrence in the merely backward.

To some extent, the preliminary results of the study of children with retarded speech development in Edinburgh have demonstrated that there is a close correlation between the extent to which speech development is retarded and the extent to which the ability to learn to read and to spell is impaired. It is apparent, however, that there are numerous causes of failure to read and spell. Such environmental factors as frequent changes of school, inappropriate and inadequate schooling, frequent absences from school, mental defect, deficiencies of hearing, serious abnormalities of vision, and combinations of these all contribute to backwardness in learning to read and spell. But in a high proportion of children who are slow to learn to read and write–in spite of the fact that they are of average intelligence, come from stable homes, and have experienced "normal" educational experiences–other explanations of their educational disabilities must be sought.

A variety of clinical syndromes have been described that may be recognized in children of average intelligence without gross physical handicap. Some of these are associated with recognizable neurologic abnormalities, as in the case of the "choreoid syndrome" of Prechtl, in children suffering from mild ataxic cerebral palsy, or in those with other indications of "minimal cerebral dysfunction." In many of these patients, it is found on close examination that educational disabilities are not confined to reading and writing, but extend to other subjects, including arithmetic.[74] In patients with "minimal brain dysfunction," there are likely to be considerable variations in the degrees of visuospatial or audiophonic handicap suffered by different patients.

Many patients who show serious specific retardation in the ability to learn to read and write do not have any indications of brain damage. Many, although not all, have a family history of retarded development in learning to speak or to read and write. In these patients, difficulties in learning to read and write may be largely visuospatial or audiophonic, or both types of difficulties may be demonstrated in test and learning situations.

A number of different clinical syndromes of "specific" learning disability may be defined. Some are predominantly "visuospatial" in type, others predominantly "audiophonic," and others a combination. Some patients in each category may exhibit weak lateralization of handedness and "eyedness," clumsiness, or a family history of retarded development of speech and of learning to read and write.

The definition of the many syndromes that constitute "specific developmental dyslexia" or "specific dyslexia" is only now becoming possible. Fuller definition is important not only for academic purposes, but also for remedial education, so that teachers may devise appropriate programs of teaching for their pupils.

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ARCHIE A. SILVER / ROSA A. HAGIN

Visual Perception in Children with Reading Disabilities

This presentation discusses two questions: What defects in visual perception are associated with delayed acquisition of reading in childhood? What happens to those defects as the child grows into adulthood?

Our own investigations—dealing primarily but not exclusively with children who were 7–12 years old, who had full-scale intelligence quotients of at least 85 on the Wechsler Intelligence Scale for Children, and whose acquisition of reading was delayed—have led us to recognize a syndrome in which a basic component appears to be disorientation in space and in time. This disorientation is reflected in specific temporal and spatial distortions in the visual, auditory, and kinesthetic–tactile perceptual modalities and in general body-image orientation. For example, temporal sequencing of sound may be defective. In tactile and body-image perception, there may be impairment in spatial orientation of right and left, up and down. There may be specific visual defects in discrimination, in figure–background perception, in visual–motor function, and in visual memory.

Individual variations in perceptual defects charted in a perceptual profile graphically illustrate that all combinations of defects may be found clinically. Visual defects, therefore, constitute only one area of difficulty in a broad spectrum of perceptual defects that is different in distribution and severity for each child.

Perceptual investigation, too, is but one aspect of the total evaluation of the child with a reading disability. In the last year, we have examined 50 first-graders who were sent to us because their language skills were significantly below those of their peers in age, intelligence, and educational exposure. We found a very mixed etiologic group. In such a group, approximately one third will have the syndrome described (specific perceptual defects), immature postural responses, and clinical evidence that clear-cut cerebral dominance for language is not established. Another one third will have this basic syndrome, but, in addition, neurologic abnormalities in one or more areas: in muscle tone, power, and synergy; in cranial nerves; in deep, superficial, or pathologic reflexes; or in impulse control and autonomic lability. Of the remaining one third, an occasional child (two in our sample) will have a peripheral sensory defect. Four children were diagnosed as schizophrenic. One was found to be immature in all aspects of development and functioning at a defective level on intelligence testing. In three, emotional problems were considered to be the cause of language retardation, and in four children perceptual deprivation at critical ages was considered causal. Because of these complicated etiologic factors, we believe that evaluation of the child with a reading disability requires, at the very least, neurologic, psychiatric, psychologic, and educational examinations.

We are concerned here with children whose language skills, as measured by standard tests, are below those expected from their intelligence and educational experience and whose peripheral sensory apparatus is intact. The defects in visual perception that are associated with delayed acquisition of reading include defects in visual discrimination, in visual–motor ability, and in visual memory. Although it may be difficult to isolate them, attempts to do so should be made, because they may reflect different aspects of brain function. In visual–motor dysfunction, for example, the critical defect may lie in the praxic element, not the discrimination one; and in defects in visual memory, complex association or cognitive factors may be involved, and not discrimination or praxis. Practically, these areas of visual perception may be studied by tasks of matching and recognition, of copying, and of recall, singly and in combination.

VISUAL DISCRIMINATION

The outstanding defect in visual discrimination is in orientation of a figure in space. Orientation may be affected not only in the right–left, or vertical, plane, but in the horizontal plane, in the depth axis, or in various degrees of clockwise or counterclockwise rotation.

The general problem of axial rotation was studied by Wechsler and Pignatelli[35] as early as 1937. The ability to orient a figure in space correctly in all axes is not acquired until the age of 5, 6, or 7 years, depending on the test used. Garvey and Herman, working in our clinic, examined the perceptual maturation of 50 children, 4.5–5.5 years old, who made up the total population of a "normal" Head Start nursery. All the children easily recognized cylinders, rectangles, wedges, squares, and columns when the forms were presented in three or two dimensions. When, however, the wedge was presented in two dimensions in various spatial orientations, 19 of the 50 chose an incorrect orientation. How many of those 19 will have difficulty in learning to read or in some aspect of language development would be important to follow. A partial answer to this question is suggested by a study by Wechsler and Hagin,[34] who investigated the problem of axial rotation in 50 first-grade children and 50 third-grade children in a middle-class suburban school. A portion of their study concerned the matching or visual discrimination of an asymmetric figure, shaped roughly like a lamb chop, imprinted in eight different positions representing the lamb chop in various axial rotations. Their data indicate that children who most frequently perceive the figure in its correct orientation show a higher degree of reading readiness, as measured by conventional tests, and make more rapid progress in beginning reading. Furthermore, there is maturation in this discrimination function from the first to the third grade: the frequency of rotational errors on recognition drops from a high of 13% in the first grade to 3% in the third grade. In short, a significant proportion of children who continue to have problems of visual spatial orientation will have difficulty in the acquisition of reading skills.

We have further investigated the problem of visual spatial orientation in older children by using the flag figure and the recognition of asymmetric matrices and overlapping forms. The flag figure consists of two crossed diagonal lines with a square drawn at the end of each, having the diagonal line as its base. In effect, it is a picture of four crossed flags. The flags are oriented in various directions; a 7-year-old child of average intelligence can easily recognize the flags in different orientations, but a child with a reading disability will have difficulty. Similarly, asymmetric matrix-like forms and complex overlapping forms present a problem in recognition. Of 60 children with reading disabilities who were involved in a perceptual training project over the last two years, only ten were initially able to recognize and match simple, asymmetric, and complex forms.

In our experience, 80% of children with reading disabilities have difficulty with the orientation of visual stimuli in space. This is at a level of visual recognition and does not involve verbal symbols.

Spatial orientation is further confused in the child with a reading disability, when a figure–background problem is introduced. To minimize the influence of the motor component, figure–background problems may be studied by recognition of pictures, hidden within a background by a variety of techniques.[18,23,32,33] Our own preference for children 8–11 years old has been the marble board. This test, adapted from Werner and Strauss,[36] has been standardized by Crain and Werner.[4] It uses identical square boards, each having 10 rows of 10 depressions. The depressions are painted black, the remainder of the board gray. Standard designs are formed with black marbles in the depressions, and the subject is asked to reproduce the design with marbles on a second board. This test eliminates a verbal factor present in the hidden images, and becomes a problem in visually distinguishing the orientation of the test pattern. It reduces but does not eliminate a motor factor, in that the marbles must be placed in the depressions to make the pattern. Of children with reading disabilities, 50% will have difficulty with this task. Their errors appear to involve spatial orientation and can be categorized into the following groups: (1) difficulty in constructing diagonals and angles, (2) omission of marbles in the figure, (3) displacement of the figure or its parts, and (4) the use of tactile clues, such as fingering the depressions. That these errors are not praxic is shown if a color contrast (e.g., using red marbles) enables the child to reproduce the figure correctly.

The ability to clearly distinguish a figure from the background may be examined by means of ambiguous figures, such as the familiar vase–face (Rubin's goblet figure) illusion. Normally, awareness of the figure or background will alternate—at one time the profile and at another time the vase will be the foreground. Why this fluctuation occurs is not known.[24] The number of apparent changes per minute, however, may be determined. The frequency of apparent change decreases with age and with structural damage to the central nervous system. In children with reading disabilities, a bimodal curve is obtained, at one end of which are those with extremely rapid fluctuations (20–30 per minute), and at the other, those with extremely slow fluctuations (4–6 per minute). The significance of this test is unknown, and the test itself, relying on subjective reporting by the child, may not be reliable.

On the basis of having studied their discrimination of geometric forms and their errors in figure–background tests, we may postulate that, symptomatically, a significant defect in the visual perception of children with reading disabilities lies in defective ability to orient a figure in space correctly. This defect does not necessarily imply structural damage to the brain, but it does suggest a lag in the maturation of some brain function. This hypothesis may be investigated further by tachistoscopic methods; the emergence of primitive patterns of visual discrimination may be observed in normal children as the exposure time is successively reduced.[27] These primitive patterns are similar to those which children with reading disabilities will display with unlimited exposure time.

VISUAL–MOTOR FUNCTION

Visual–motor function adds the functions of fine motor coordination and of praxis to the capacity for precise visual discrimination. Even if tests of visual discrimination are normal and there are no defects in fine motor coordination, visual–motor function may be disturbed because of a defect in the ability to imitate motor patterns. Frequently, a child can recognize that his copy of a pattern is incorrect, but he cannot make it correspond. Praxis, like any other function, has a sequence of maturation, with the primitive visual–motor pattern proceeding from scribbles to connected loops drawn clockwise in a horizontal row, to single closed loops, to crossed horizontal and vertical lines, to the square, to the triangle, and finally to the diamond at age 7.[27] Crossed diagonal lines require approximately 8 years for maturation.[1] Delay in the maturation of visual–motor function has been found by de Hirsch *et al.*,[5] Goins,[8] and Monroe[21] to correlate significantly with the presence of reading disability.

Our own observations of children with reading disabilities reveal that, on the Bender-Gestalt test, they perform below their expected age and intelligence. Significant defects are indicated by four characteristics: difficulty with angulation, either immature or actually stellate; a tendency to verticalize the diagonals, and to rotate entire figures toward the vertical; replacement of dots with primitive loops; and use of cues, such as the edge of the paper or a previously drawn figure. These findings consistently appear, in greater or lesser degrees, in approximately 90% of children with reading disabilities. The findings agree with those of Keogh.[13]

There are, of course, many other ways of testing visual-motor function—for example, the copying of geometric figures other than the Gestalt form[8,21]; the parallelogram test of Luria,[18] in which the patient is to place a circle in the appropriate corner of a parallelogram to reproduce a given parallelogram and circle, all presented in various spatial orientations; the Kohs block test[10] or the block-design subtest of the Wechsler Intelligence Scale for Children; and Raven's progressive matrices.[25] The latter example, however, may involve cognitive processes other than visual-motor function. Such tests as the Gottschaldt figures, used more for adults,[11] combine a figure-background problem with a visual-motor one. The defects in visual-motor function found in children with reading disabilities, like defects in visual discrimination, may be interpreted as being related to spatial orientation. These defects are characterized by performance on a lower level of the maturational scale than expected from the age and intelligence of the child.

DEFECTS IN VISUAL MEMORY

Tests of recall of visual stimuli appear in most surveys of visual perception in children with reading disabilities. Monroe,[21] for example, required children to draw from memory 16 designs that were presented in groups of four for 10 sec/group. In a portion of the Wechsler and Hagin study[34] cited earlier, recall of the stimulus card exposed for 3 sec and then removed from sight proved to be more difficult than visual discrimination, and a poor score in recall was significantly related to poor reading in both first- and third-graders. The Benton visual retention test,[2] however, as reported in 20 children aged 9–11 years with reading disabilities, revealed only two children with defective performance.[16]

Our own observations indicated that, when recall involved the memory of asymmetric figures or the memory of a visual sequence, problems arose. The normal 6-year-old, for example, can remember a series of five small and large square blocks arranged in a sequential pattern; if the blocks are removed, he can replace them in their correct sequence. The 5-year-old has difficulty with this, and when the square blocks are replaced by wedges with differing orientation, even the 6-year-old will falter.

Our children with reading disabilities cannot draw from memory the complex asymmetric figures or sequences of overlapping forms that they have just seen for as long as 15 sec. Whether this is a problem of visual memory or a reflection of the visual–motor problem cannot always be determined from this test alone but becomes apparent in the context of a total examination.

Problems in visual memory in children with reading disabilities may be interpreted primarily as defects in spatial orientation. This defect is also seen in visual discrimination, in visual figure–ground perception, and in visual–motor functioning. The visual discrimination function may be said to be immature if there are problems in orienting a figure in space or discriminating a figure against the intruding background. Visual–motor function may be said to be immature if there is a tendency to revert to primitive verticalization and spatial confusion.

That these are difficult problems can be seen in efforts to correct them through training. Of 60 children involved in a perceptual-training project, 38 required more than 10 hr and three required more than 25 hr of training to learn to recognize, copy, and recall even the supposedly simple geometric forms; the total length of individual training sessions required to progress from simple forms to complex forms reached well over 50 hr for each child.

What happens to these defects? As the child grows into adulthood, these perceptual areas do show some evidence of maturation. The defects do not, however, completely disappear, and some evidence of reading disability persists even if there is adequate academic, social, and vocational progress.[26] A follow-up study of 24 children with reading disabilities originally studied and treated in our clinic between 1949 and 1951 and studied again as young adults in 1961 and 1962, some 10–12 years later, revealed the persistent stamp of reading disability.[29]

In visual–motor testing, for example, the Bender-Gestalt drawing does show statistically significant improvement in rotation and verticalization and in ability to cross the midline. Angulation difficulties, however, persist, and although the degree of rotation is decreased, some slight primitive tendency toward verticalization remains. When contrasted with a control group of adequate readers from a similar clinical population, these primitive tendencies in those who had reading disabilities as children are seen more clearly.

In the area of figure–background perception, those with reading disabilities in childhood made significantly more errors than did controls in reproducing diagonals, in omitting marbles, in displacement of the figures, and in use of tactile or color cues. Twelve years later, significant improvement was noted in only one area: omissions of marbles from the figure. Although improved, the adult with a reading disability still suffered from a figure–background problem, because he had not attained the maturation level of the control groups.

If we analyze our data in terms of adult reading achievement, we find that the adequate readers tend to have been less severely retarded in reading when tutoring was initiated, that as children they had fewer perceptual problems, and that as children they had a significantly greater proportion of verticalization and rotation errors in visual–motor performance, but significantly fewer figure–background problems. Diagnostically, there were fewer "soft" neurologic signs (i.e., signs of minimal brain damage).[29]

Other investigators have considered the effect of training in perception on various aspects of reading behavior. Among these are Levin and Watson,[17] Staats *et al.*,[31] Monsees,[22] Elkind *et al.*,[6] Budoff and Quinlan,[3] and McNeil and Stone.[20] The persistence of perceptual defects into adulthood, however, led us to consider the possibility of a direct attack on perceptual defects in childhood. Instead of circumventing them, we would try to train them out. Accordingly, we devised training techniques to correct the specific perceptual defects found in careful evaluation of each child. In the visual area, this consisted primarily of the recognition, copying, and recall of simple and asymmetric, matrix-like, and overlapping forms, exercises in the orientation of forms in space, and training in the isolation of the figure from the background. Details of this experiment may be found in Silver *et al.*[30]

After 50 sessions of perceptual training, the overall Koppitz[15] scores on Bender-Gestalt testing are improved significantly at the 0.001 confidence level (Wilcoxon test).[28] Examination of the error characteristics reveals improvement in angulation, verticalization, rotation, accuracy of joining, and ability to change direction in card 6, all at the 0.005 level of confidence, using the chi-square test. On the marble–board test of figure–background perception, the total score, using Goldenberg's system,[9] is significantly improved (at the 0.001 level). Individual error characteristics show significant improvement in omission of marbles, angulation errors, and diagonal errors at the 0.0005 level and in displacements at the 0.005 level. These improvements are not made after control sessions. It appears, therefore, that specific training of visual discrimination, visual–motor copying, and visual memory can indeed result in improved ability in these functions. Is this reflected in improved reading ability? Of 58 children with reading disabilities treated in our program of perceptual correction, 43 improved in Koppitz scores and 15 did not. Oral reading and reading comprehension improved significantly in the children with improved Koppitz scores; improvement was not significant in the 15 whose Koppitz scores did not improve.

In summary, the evaluation of visual perception is but one part of the perceptual examination of the child with a reading disability. Perceptual study is, in itself, only one part of the neurologic, psychiatric, psychologic, and educational evaluation considered minimal for the understanding of such a child.

Defects in visual perception are, however, associated with delayed acquisition of reading. In visual discrimination, the outstanding defect is in orientation of a figure in space and in clearly discriminating a foreground figure against an intruding background. In visual–motor function, the main defect is immaturity in ability to reproduce a correct spatial orientation of a figure, and specific immaturities appear in the retention of a primitive tendency to verticalize figures and in difficulty with angulation. Defective visual memory shows up in the difficulties in spatial orientation and memory of sequences of visual forms.

As the child grows into adulthood, these defects tend to lessen; but usually they do not completely disappear, and some evidence of dysfunction remains.

Visual perceptual defects in childhood may be reduced by training procedures that use direct perceptual stimulation. Improvement in perception appears to be associated with improvement in oral reading and in reading comprehension.

*CEREBRAL DOMINANCE**

The functional relationship between the two cerebral hemispheres has been a focus of our attention in attempting to understand the problems of children suffering from developmental (specific) reading disability and those with minimal neurologic signs.

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*This section was added after the conference.

If we analyze the perceptual defects found in children with reading disabilities, we are impressed that in each perceptual area the defect may be interpreted as a defect in spatial or temporal orientation. This defect appears in the visual, auditory, and haptic avenues of perception, and in the total body-image concept, where problems of right–left orientation are evident. If we attempt to carry our understanding of these perceptual defects back one step further, we suggest that defects in spatial and temporal orientation may in themselves be caused by a lack of clear-cut cerebral dominance. The finding of Sperry (see Sperry's presentation, p. 167) that the function of visual spatial orientation resides primarily in the lesser hemisphere, suggests that these children with reading disabilities have functional defects in the lesser hemisphere or in its relative balance with the dominant one.

Suggestive evidence that the problem of cerebral dominance is important in reading disability has been advanced by McFie[19] (phi phenomena) and Kimura[14] (dichotic auditory stimulation) and affirmed by our observations with the extension test of Hoff and Schilder.[12] On the latter test, children with reading disabilities do not have clear-cut cerebral dominance.

To be consistent with our hypothesis, then, the teaching of reading should theoretically begin with the establishment of clear-cut cerebral dominance for language. We do not know how to do this, but we have attempted to teach reading to children with reading disabilities by working with the next step in their neurophysiologic maturation—namely, by improving the accuracy of their perceptual input.

The object of this experiment was threefold: to determine whether we can reduce perceptual errors in children of school age by training, to ask what effect this training has on oral reading and on reading comprehension, and to ask what effect this training has on our measures of cerebral dominance.

As indicated, errors can be reduced by training; children then do better in oral reading and in reading comprehension. Also, in children in whom perceptual training is successful, tests for cerebral dominance change in the direction of more definite establishment of a dominant cerebral hemisphere. Our experiments in perceptual training were designed to give the children a firm perceptual basis for the later development of language skills.

Perhaps we should go back even further and attempt to develop cerebral dominance in the infant. If, as Gesell has suggested,[7] cerebral dominance is based on tonic neck reflexes, an infant who does not exhibit a preferred reflex direction is prone to develop language disability. Our efforts, using Dr. Lipsitt's techniques (see p. 381), may well be to establish a preferred direction of tonic neck reflexes in the first year of life and thus enhance very early the perceptual pattern of functional asymmetry.

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RICHARD L. MASLAND

Implications for Therapy

CLASSIFICATION

Dr. Ingram (p. 405) has provided two types of classification. The first is etiologic; it includes organic disease, deviation of development, and less well-defined and more heterogeneous problems. The second is functional; breakdown in the mechanisms involved may occur in any of several crucial stages of the process of learning to read. He has postulated three points of possible breakdown in this process: a failure of perception, an inadequate language function, and a difficulty (or inability) in associating a visual symbol with the sound that it represents.

Language Function

Dr. Ingram has emphasized the linguistic element in reading—the importance of the pre-existence of an adequately developed language function in the child. His demonstration, also shown by Dr. Hirsh (see p. 231), of the inconstancy or lack of precision in the use of language at the time the child is expected to be learning to read is an important contribution to our understanding of the problem of the child at this age. Although Dr. Ingram recognizes the frequency of other factors in dyslexia, his prospective study documents clearly the relationship between deviations in language development and deviations in learning to read. Drs. Silver and Hagin, in the preceding paper, also recognize the diversity of the problem, but they emphasize the significance of disorders of perception. I have tried to relate these learning-to-read processes in a neurophysiologic and neuroanatomic model that I think has some value for understanding the complexity of the process with which we are dealing and the various points of weakness at which a breakdown may occur.

Anatomic Model

In regard to the language function, the indications are that the centers for spoken language develop in the left hemisphere. Although considerable plasticity still exists in the child, it is reasonable to assume that the analysis and use of auditory language, by the time a child enters school, are centered largely in the left hemisphere.

Dr. Sperry (p. 167) has indicated that the appreciation, analysis, and recognition of geometric patterns and the orientation of self and objects in space constitute a function that, at least in the adult, is most skillfully carried out in the right hemisphere, particularly in the parietal region. At some point in the reading process, it is essential that there be established an association or a relationship between the visual pattern of a letter (which is, according to this concept, most effectively analyzed in the right hemisphere) and the meaning and sound of that letter, whose analysis is mediated in the left hemisphere. Experimental work[3] strongly indicates that such interhemispheric associations are much more difficult to make than intrahemispheric associations.

Teleologically, the visual process—the spatial orientation function—must be concentrated in a single hemisphere. As was emphasized by Dr. Sperry, this appears to be a very strongly unilateral function. The pigeon's eyes are on the sides of its head, and it has no stereoscopic vision: there is no uncrossed visual pathway in the visual tracts (Figure 1). If you cover one of the pigeon's eyes, teach it to discriminate between mirror-image objects with one eye, and reward it for selecting, say, the "b," and then cover that eye and permit the bird to attempt the task with the other eye, it will select the mirror image.[1] The same experiment has been done in the monkey (Figure 2). If the optic chiasm is sectioned, the animal will have only a unilateral input to each occiput. Exactly the same results are observed in such a monkey that is first trained with one eye and then tested with the other.[4]

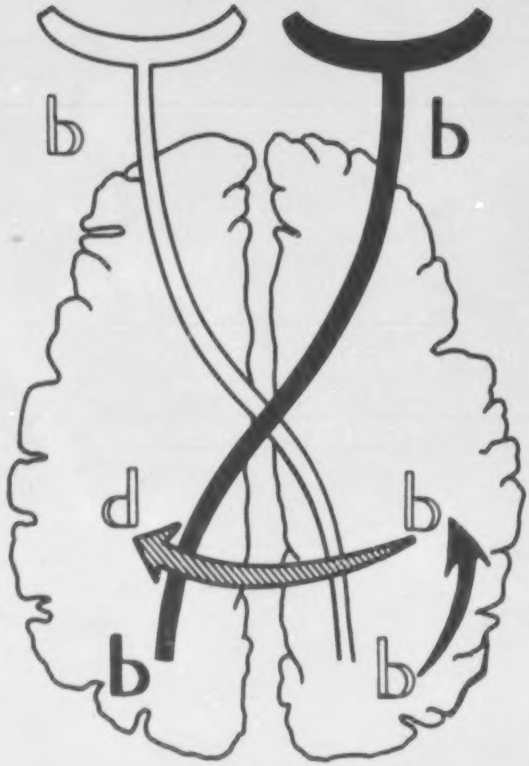

FIGURE 1 Interhemispheric transfer in the pigeon. A bird trained to peck the letter "b" with its left eye has the trace of this image relayed to the left hemisphere as "d." If it is then required to select between "b" and "d" with its right eye, the new image in the left occiput is compared with the reversed image of the memory trace in the left hemisphere, and the mirror image, "d," is selected. (After Mello.[1]) (Reversals in the retina are disregarded for simplification.)

Returning to the pigeon, how does one explain this phenomenon? If the animal is trained with the left eye, it becomes responsive to an image projected to its right occiput. This image, presumably, is similarly located in the visual association area, after which it is carried by a point-to-point mechanism as a mirror image to the other hemisphere. If the animal then observes the object with its right eye, the object that matches is, in fact, the reverse object, and not the original image.

Integration Deficit

From this experiment, it would appear that under ordinary circumstances in the human there must exist in each hemisphere two conflicting mirror images—one representing the direct visual input to that

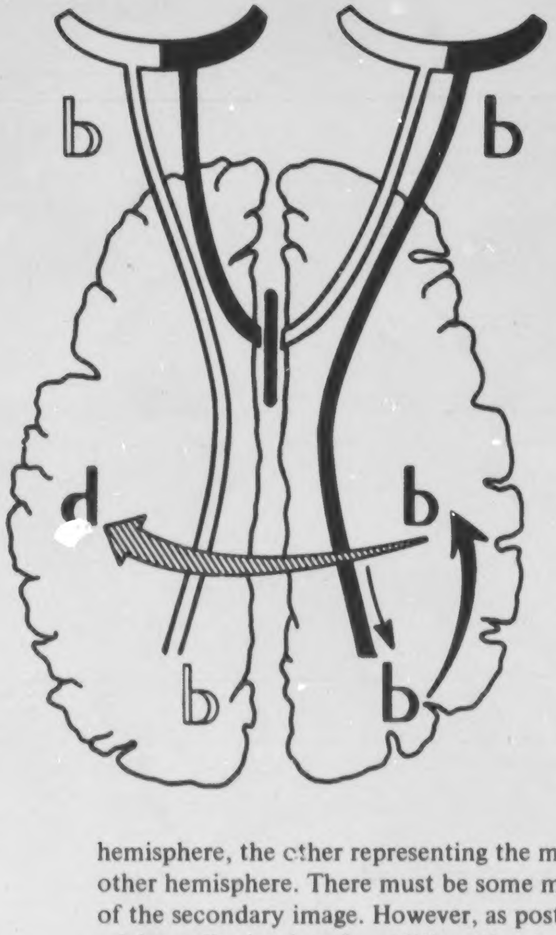

FIGURE 2 Interhemispheric transfer in the monkey. If the optic chiasm of the monkey is cut, each eye will project only to the ipsilateral hemisphere. "b" seen with the right eye is projected only to the right hemisphere and relayed to the left hemisphere as "d." If the monkey is then required to use its left eye to select between "b" and "d," it will select "d," because it corresponds to the memory trace of the left hemisphere. (After Noble.[4]) (Reversals in the retina are disregarded for simplification.)

hemisphere, the other representing the mirror image relayed from the other hemisphere. There must be some mechanism for the suppression of the secondary image. However, as postulated by Orton,[5] confusion might easily develop. In addition, the studies cited above suggest that the most effective integration of complex visual patterns is in the right hemisphere. There must be a consistent reversal whenever images are associated with auditory language symbols mediated in the left hemisphere.

Perceptual Deficits

The convincing demonstration by Drs. Silver and Hagin of the nature of the perceptual deficits of their group of children lends some support to the thesis that disturbance of the mechanism for suppression of the secondary image underlies much of the children's disability. For example, they pointed out the tendency toward verticalization. Consider the problem of the child who must distinguish between a vertical line and a horizontal line. That is simple, because the vertical line is exactly the same in the two hemispheres (as is the horizontal line). But consider the child who must distinguish between two diagonal lines that are, incidentally, also at a 90-deg angle. Theoretically, they might be no more difficult to distinguish than the horizontal and vertical lines. In this instance, the lines are confused in the two hemispheres and the child has to remember not only the appearance of the line but which hemisphere he is seeing it with. It is evident from Silver and Hagin's studies that there may be a very sound reason to assign a neurologic basis for the perceptual difficulties of these children. In respect to recognition of figures and symbols that are to be used for communication, in which an association must be established between the visual image and the language function, the child is being required, at first, to use two hemispheres, and an unusually difficult task is being imposed on him.

Milner's data[2] suggest that, in the adult, although visual pattern recognition is still most effectively mediated in the right hemisphere, the recognition of symbols is most effectively accomplished by the left hemisphere. This suggests that the problem of establishing association between vision and language function is handled by having the center for symbol recognition develop in the left hemisphere, rather than in the right, where pattern recognition is ordinarily most effectively handled.

Permanence

Drs. Silver and Hagin have pointed out that these perceptual deficits are frequently permanent. There is, however, a tendency to speak of them as developmental lags. Although many children may learn to overcome their disabilities, for many, the disabilities nonetheless remain. Rawson[6] has documented that, although her group of dyslexic boys managed to circumvent their disabilities and become effective readers, they were still different from average adults. They continued to have spelling difficulties, and the perceptual deficits sometimes persisted.

We are talking about at least two different tasks when we speak of "reading disability": the task of the third-grader, who has to be able to read words, probably requires a concentration on pattern recognition and precision of pattern recognition; and the task of the high-school student, who has to extract meaning with a minimum of cues, requires a different type of skill even though he must first have mastered the earlier task to some extent if he is to accomplish this one. There is much to suggest that the bright child, even if he continues to have a very serious perceptual deficit, manages to become a reasonably effective reader by using sketchy cues, which are all that is essential if one is able to extrapolate, as was discussed in this meeting.

TRAINING GOALS

I have some reservations about the hypothesis presented by Drs. Silver and Hagin that, to teach a child to read, one should address oneself to the underlying deficit in perception. There is a question of whether to teach to the deficit or around the deficit. The primary task required of the child is the recognition of a letter or word and its association with a sound or meaning.

The teaching of perceptual skill is not an end in itself; we are not interested in training the child simply to perform well on the Bender-Gestalt test. The goal is the recognition of letters. Why not conduct perceptual training with the very objects or patterns that must ultimately be learned? For some, this may be aided by training and practice in distinguishing right from left. For others, haptic sensations may be useful to supplement weak or confused visual skills. For others, explanation, description, and logical analysis of letter shapes may be helpful. For still others, the use of a name or sound as a label for the symbol may be helpful. Earlier at this meeting (see presentation by Hirsh, p. 231), experimental evidence has been reported that indicates that a letter or trigram invested with a name is more readily recalled than one without a name.

As to experimental evidence of the value of perceptual training, I know of only one adequately controlled study: that of Rosen.[7] He investigated early readers in 25 classrooms; half the children were given a half-hour of perceptual training each day, and the other half were given a half-hour of extra reading instruction. At the end of a semester, they were all tested for perceptual motor skill and reading. The children who had received the extra training in perceptual motor skills were better in perceptual motor skills; the children who had received the extra training in reading were better in reading. The results were the same when the classes were broken down into three equal groups of good, medium, and poor readers. There was, however, a small subgroup of 50 very handicapped boys in whom perceptual motor training seemed to offer some advantage, but the results were not statistically significant.

IMPLICATIONS

This summary of some of the possible neurologic, perceptual, and language-function deficits that may come into play in dyslexia leads to an important thesis: There is no panacea for dyslexia, and we are dealing with a wide diversity of problems and certainly with a high degree of individualization. In addition, the importance of early experience in the development of intellectual abilities must be emphasized. It is still unclear in which of these children the disability has an organic or structural basis, and in which an environmental or experiential factor is responsible. The types of perceptual motor disability that Drs. Silver and Hagin defined are reportedly more common in the underprivileged segments of our society than among other groups. Is that because these children are more likely to be organically damaged? Is it because of unfavorable social and environmental experiences? Or is it because of a combination of the two? Greater attention must be paid to the specific developmental experiences related to the establishment of perceptual skills and to the language abilities that are essential in learning to read.

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MANAGEMENT OF CHILDREN WITH PERCEPTUAL AND READING DISABILITIES

H. BURTT RICHARDSON, JR.

Relationship of Research to Health and Educational Services

Research in the basic sciences has a long history of direct influence on developments in both health care and education. Recent decades have brought a tremendous upturn in basic scientific investigation, much of which has focused on new areas with high relevance to the learning process. Scientific discoveries of the last century have influenced these areas, as well as society itself, in two general ways. First, a number of basic scientific discoveries of elemental significance have produced profound changes in scientific viewpoints. Second, and more frequently, science has been called on to provide the answers to specific practical questions raised by the institutions of society. In this latter manner, both the health and the educational service systems have grown to rely increasingly on basic scientific research to solve some pressing problems.

PEDIATRIC PROGRESS

For a variety of reasons, the scientific bases of educational practice and medical practice have remained divorced from each other. Medicine, over at least the last half-century, has relied increasingly on physiologic, chemical, and pharmacologic research. Dramatic changes in health care have resulted from research discoveries and technical developments in both diagnosis and therapeutics. These have led to the solution of many of the most pressing problems that faced medicine fifty years ago. Over the same period, however, a broader concept of health care as a community responsibility has become widely accepted. Particularly in the area of child health care, an entirely new set of responsibilities has emerged because of the solution of such massive child-health problems as infectious and diarrheal diseases. The responsibilities of the primary physician caring for children and the health-services team collaborating with him are increasingly focused on guidance and modification of early developmental processes within physical, psychologic, and social spheres during infancy and early childhood. Much observational information on developmental processes, particularly during infancy, has accumulated over the last several decades within a pediatric context, but there has remained considerable isolation between the application of this information and the application of information from simultaneous research in other areas of the behavioral sciences.

PEDAGOGIC PROGRESS

The field of education has made decisions based on the results of investigations in different areas of research. Education has, of course, been strikingly influenced by basic biologic discoveries, but in large part the investigational procedures in education have involved empiric problem-solving studies and often group evaluations. There is now increasing interest in the educational processes of individual children and, in particular, in relationships between behavioral patterns noted in the educational setting and descriptions arising from neurophysiologic and neuroanatomic research. The perspective of educational responsibility, heretofore limited to the traditional school years, is also undergoing considerable reappraisal. As educators become increasingly concerned with the practical importance of early experience and as they participate in such programs as Head Start and special nursery schools, the health and educational services involved form an inescapable relationship. Research within both broad areas is beginning to overlap enough that complementary, if not collaborative, services appear to be increasingly possible.

FUSION OF BIOLOGY AND BEHAVIOR

One manner of categorizing research areas called on to elucidate the learning processes of early childhood might be to divide them into broad biologic- and behavioral-science groups, recognizing, of course, the essential overlapping in application to individual children or populations. The biologic approaches have included neurostructural, neurophysiologic, and neurochemical research. Those behavioral sciences which have provided the greatest progress in medical and educational thought have included psychodynamics, perception, cognition, and learning theory. It is well to recognize that behavior is the measured endpoint or correlate of most of these scientific studies, regardless of their theoretical basis; but the behavior described in most studies of child development has occurred in the natural environment, rather than in the controlled experimental setting. Child development has long been associated with either the biologic or the psychodynamic approach because of their common connection with medicine. A better approach to developmental studies as a mode of testing any scientific hypothesis across time resides in the rapidly growing young organism.

MAGNITUDE OF HEALTH-CARE DELIVERY PROBLEM

The current commitment to providing the best possible health care and educational opportunity to the entire child population carries with it an obligation to quantitate the problem, as well as to appraise the system by which the services are delivered. "Dyslexia" is a descriptive term with different definitions, all of which indicate handicaps in learning to read. Dyslexia is only one of various learning aberrations that impede classroom success. If dyslexia implies reading skills 2 years behind grade level, an estimated 15% of children are dyslexic.[3] Even on the basis of a neurologic definition, such as that by Critchley,[1] who considers dyslexia a "very real, organic problem" representing a "specialized instance of cerebral immaturity," a prevalence of 10% is likely. The inclusion of other categories of learning disorders, of course, makes the number of children involved much higher. It is clear that the appropriate management of learning disorders of such prevalence must fall in large part to both primary health-care facilities and what we might consider the primary educator, the classroom teacher.

Sharing of Influence

If we assume that a child's learning depends on environmental variables (and available evidence leaves little doubt of that), the primary influences on a child's educational success are his parents and later his classroom teacher. It is unreasonable to plan effective modification of environmental stimuli for a proportion of the population as high as, say, 20% without developing the requisite manpower resources. Traditionally, parents and teachers have created learning conditions for children relatively independently. A parent's decisions concerning the presentation of stimuli depend on cultural norms, intuition, and guidance (particularly in the years of infancy) from health-services personnel. A teacher's decisions depend, in addition, on professional training and the professional expectations of the school itself. A supplementary diagnostic staff in the school frequently operates less to modify the classroom teacher's decisions than to make independent administrative decisions regarding class placement.

Transmission of Information

Since a highly relevant portion of a child's exposure to learning conditions is under the control of the parents, it is worth exploring points of contact through which information can be transmitted to them. General information can be transmitted, of course, through the usual communication media of the culture, including the parents' formal education; but specific guidance concerning any single child, particularly one in whom a learning problem is evident during the preschool years, has generally fallen to a member of the primary health-services system. Within both pediatrics and nursing, the two professional groups delivering primary health services to the majority of American children, anticipatory guidance and behavioral counseling are common and well-accepted practices that are included with the provision of adequate comprehensive health care. The contact points for the acquisition and transmission of relevant information concerning the learning needs of the individual child exist, therefore, within the primary health-services system, and the personnel in that system are committed to the principle of appropriate guidance of parents. What is lacking is an informational system that might provide the communicative interface between parent, teacher, and primary health-service personnel. It would appear, then, that primary health-service personnel, the classroom teacher, and the mother are in the best positions to apply most effectively the results of basic behavioral-science research. These are the only persons in the societal system who have sufficient contact with children to modify significantly the environmental conditions under which children learn.

APPLYING NEW KNOWLEDGE TO DIAGNOSIS

If our intention is to provide health and educational services to all children, we must reconsider the very nature of our diagnostic and management procedures and be willing to modify those that are out of step with scientific evidence or our stated goals. In both health and education, there is a long record of excluding children from relevant experience and services on the grounds of developmental delays manifested by failure on readiness tests or other measures of general achievement. In many settings, children who score poorly on standard intelligence tests are still completely excluded from educational experiences at the lower age levels. The labels applied by many current diagnostic techniques are frequently used to separate children permanently from the success-bound educational stream. Such descriptions as "retarded," "brain-damaged," and "dyslexic" tell nothing of the mode by which a child might succeed or of the conditions under which success will be most likely. In addition, such labels are remarkably poor in communicating causative factors, a traditional (and in the case of learning disorders, frequently irrelevant) preoccupation of medicine. Labels serve only to convince the primary physicians, the classroom teacher, the parents, and, most tragically, the child that success is impossible and effort of doubtful value.

How, then, can the diagnostic process be directed so that, on the one hand, it serves our stated goals and, on the other hand, it utilizes more adequately the evidence revealed by the behavioral sciences? First, it must be closely related to or even a part of the very processes that it sets out to detect. Second, it must take into account the critical importance of the specific environmental conditions that influence the behavior observed. Third, it must recognize the dynamic factors at play in the diagnostic learning situation that may have more general applicability to learning at home or in school. Educators have long focused their interest on diagnosis of relevant functions, but until recently they have placed much less emphasis on the conditions of testing or the dynamics of behavioral change manifested in the learning process. School achievement has been successfully predicted from measures that sample behavior that correlates highly with behavior demanded in school. Behavior observed in this way is closely related to the processes of primary interest, but, as alluded to above, it tends to select for failure, not for success.

The diagnostic appraisal of conditions under which a sample behavior is noted or not noted has a much higher probability of offering clues to a child's future success in learning. The basic studies in perception, whether biologic or behavioral, offer much to the diagnostician whose obligation is to evaluate the significance of stimulus variables in a specific child. Relevant educational material may be presented to a child through a variety of stimulus modalities, revealing a pattern of function that indicates not only at which point in an educational program the child might be expected to succeed, but also through which channels of stimulus input success will be achieved most easily. Evaluation of a child's perceptual function in distinguishing signal from noise along various input channels is equally relevant in that both stimulus quality and input modality can be easily modified in the classroom or home. A relatively brief examination of a child[2] can sample skills in attending to auditory, visual, and tactile input of educationally relevant information and can appraise the interference of noise in the system. At the same time, one can gauge, in a standardized fashion, motor competence in the performance of specific school-required functions, such as speech intelligibility, handwriting, and general fine motor coordination.

In addition to appraisal of the placement in a teaching program appropriate for a child's success, the diagnostician observing a child's behavior has an excellent opportunity to evaluate dynamic elements of the learning process, i.e., the relationships of factors through time, as contrasted with the static description of stimuli or observed behavior at a single point in time. Research in learning has done much to elucidate the relationship between rates of modification of behavioral responses and the occurrence of some consequences of modifications in the environment. It is highly relevant to the educational process, whether in the home or in school, to be aware of these dynamics in a particular child. Thus, if learning tasks can be incorporated into the diagnostic measure, it is possible to appraise the rate of learning under standardized stimulus conditions. The social consequences of a child's responses are also sufficiently under the control of the examiner that a preliminary appraisal of what maintains his responses is often possible. Other dynamic factors, such as the child's ability to have postponed the social recognition of his accomplishments, can be judged in a similar setting. Such observations are very relevant to the mother and teacher with whom the child interacts; these interactions form the basis of both socialization and educational processes.

In summary, the remediation of learning disorders requires early and relevant diagnosis of deficits in the processes on which successful learning at home or in school depends. The number of children involved is so great that effective diagnostic procedures must be in the armamentarium of primary health-service personnel and classroom teachers. The development of adequate diagnostic measures derived from basic knowledge in perception, cognition, information-processing, and the dynamics of learning offers the best hope for collaborative efforts by the health-care and educational systems in the early childhood years.

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PANEL: Thomas T. S. Ingram, Barbara Keogh, John H. Meier, H. Burtt Richardson, Jr., Helen M. Robinson, Donald Shankweiler, and Francis A. Young, Moderator

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Conference Implications for Education

DR. YOUNG: The value of this conference may well depend on how successfully the diverse results and hypotheses presented here are translated into forms applicable to children who must learn to handle visual input. With the purpose of fostering the translation, a group of conference participants who are working in areas that directly involve children were asked to summarize the implications of the conference.

DR. ROBINSON: For more than 30 years, I have been concerned with the problem of research in reading, the practical problem of diagnosis and correction of reading disability, and the problem of trying to help teachers understand and improve their pupils' reading. I know less today about the process of reading than I did when I began. I think that is because research in the field of reading is in its infancy. Sometimes, the research designs have not been the best. We began with what we had to work with–child behavior–which was examined in the early years primarily in relation to reading itself. Many of the studies have been comparative studies to locate areas in which there were differences between good and poor readers. That is the logical beginning, I believe, because it can give us leads as to where we might look for a deficit.

We have been handicapped, in most instances, because we have had to look for symptoms of difficulties that distinguish good and poor readers. These symptoms are inconsistent from one group of children to another or one observer to another. As a result, we have had, and still have, differences in interpretation of the results of inconsistent studies. What we have not had, I think, is consistent study of the basis of symptoms. This conference has supported the need for good interdisciplinary research in which those with the most knowledge in the related disciplines bring their knowledge to bear to help us explain the symptoms that we see.

But we need to be very clear about these symptoms. The symptoms that have been clearly delineated by Dr. Ingram, Dr. Silver, and many others lie in the areas of visual perception, auditory perception, language, and their association in such a way that reading can take place. To the extent that we can work with the investigators who are probing behind these symptoms, I think we will be able to explain what underlies the symptoms, rather than merely treating them.

Furthermore, this conference has given me, at least, some important notions in relation to techniques of investigation. The process of reading, which seems very simple to a competent adult reader, is exceedingly difficult for a child or an adult who cannot read. We need techniques to investigate the structure and function of the brain, visual and auditory input systems, language, motivation for learning, and ways in which deficits impede learning. All the physical, psychologic, and instructional information must be collated.

One thing we need to keep in mind is that the little knowledge we have now can lead to improved reading. I am reminded of a school system that I served as a consultant, in which the percentage of reading-disability cases was reduced over a 5-year period from some 20% to 5% by improving instructional techniques in the classrooms. We need to focus our attention on the 5%. I am fully in accord with the new aim of 100% success in learning to read. I hope I live to see it occur. I am not hopeful that it will happen right away, however. Essentially, interdisciplinary research takes time—time to learn to communicate, to coordinate efforts, and to study children longitudinally.

This meeting is an example of the tremendous effort it takes to absorb the detailed knowledge, the vocabulary, and the concepts of related fields. Whenever we aim toward research of this kind, we must have patience with each other; we must explain; we must aim to understand how we complement each other in our efforts.

I suggest that our deliberations after this conference be directed toward further interdisciplinary research to determine the causes of reading disability. If we can determine causes, we can make systematic diagnoses and design treatments appropriate to different causes or patterns of causes. Until we do that, we are likely to continue to rely on clinical intuition in diagnosing and eliminating symptoms. Moreover, scientific studies should lead to prevention of reading disability or to compensatory education from the beginning of schooling.

I see this conference as a beginning. Everyone here should make continued contributions to research efforts, directly or indirectly related to reading. The reading researcher needs to keep abreast of the related research, to work closely with allied disciplines, and to attempt to apply new insights. Through future experimentation, a systematic approach to prevention and treatment of reading disability should be possible.

DR. MEIER: The great problem at this conference is what I like to refer to as the "paralysis of analysis," wherein the esoteric characteristics of, say, dyslexia become relatively well delineated, and yet the classroom teacher is in the same situation today as she was in yesterday—namely, not knowing precisely what to do with a child who has failed to profit from her reading instruction.

There is a network of educational laboratories that have addressed themselves to such chronic educational problems. We have tried to determine, first of all, the incidence of learning disabilities—included among these would be dyslexia. We have started with a stratified random sample of about 2,400 children from eight Rocky Mountain states, and the incidence of dyslexia on the basis of this pilot study is, as Dr. Richardson suggested, between 15% and 20% of the regular school population. We have developed a classroom screening instrument that identifies about 80 observable behavioral symptoms, which classroom teachers have been able to use with about 94% accuracy in identifying children who, on subsequent diagnostic work-up, did demonstrate some specific learning disability. The subsequent diagnostic work-up involved 3 days of testing, including full medical and psychoeducational examinations, speech and hearing assessments, and so forth. We now are in the challenging position of attempting to go beyond the paralysis of analysis by translating the very significant work that the basic scientists are doing into both classroom practices and preschool practices. The Parent-Child Centers that Dr. Richardson mentioned, the Head Start movement, and related efforts are concerned with training parents, as well as teachers, to cope with and perhaps even prevent reading difficulties.

I am wondering what can be done in terms of early cognitive stimulation of children. Are there practical ways of proliferating dendritic spines? Do learning-disabled children have difficulties, such as Dr. Lindsley intimated, in terms of selective perception of the stimuli in the environment? Do children have ways of turning their receptors off or on? If they are turning off their receptors, are they, therefore, not growing cognitively? All these questions are of interest to us who are concerned with optimal child growth and development. We are experimenting with the crib as a learning environment, as Dr. Lipsitt suggested that it is. I see the observations reported at this conference coming together in a complex mosaic, which I think has great significance for the child who is failing to learn in school.

I would like to present a preliminary inference that we are able to draw from our data[5]—and this is a glittering generalization—namely, that nonlearning children seem to have the greatest amount of difficulty in reliably sequencing visual data in space and auditory data in time. Because of this finding, we are going to launch our major efforts toward classroom techniques that will be organized in a flowchart of approaches, giving the teacher a systematic repertoire from which to draw, to apply to specific children whose deficits may appear on evaluative protocols such as those suggested by Dr. Silver (most of which we have used). In this way, the teacher can monitor the growth of the child in response to a given educational prescription and determine whether the child can profit from that approach or whether the next one in the hierarchy should be tried.

I know this takes time and a tremendous amount of cooperation from the behavioral scientists, as well as the educational scientists (if I may call them that). I am terribly excited about the discoveries that the basic biobehavioral scientists are evidently making.

One last thing I would like to ask is: What are the optimal levels of sensory stimulation for infants and children? I think that Dr. Riesen rather hastily glossed over the notion that there may be overstimulation. We have a social issue, here, with regard to parents and teachers who may indiscriminately impose stimuli on the organism and, in fact, do damage. I gather from the informed people here that this is another area requiring considerable investigation.

DR. KEOGH: This conference has demonstrated again that there are many discrepancies in what we know about basic mechanisms in reading and that we have not related some of the more technical information pertaining to it.

For example, we have hardly begun to address the complexity of the reading task. Whether we are talking about learning disability, reading disability, or successful reading, there are, I hope, some common elements or dimensions that can be identified, and I think that these have not been defined very clearly at this conference. I don't see the child with reading disability as a different animal. He is a youngster who has particular kinds of problems in a complex-task situation, but the components of that task are the same for him as they are for the youngster who is good in reading. Therefore, I suggest that, instead of beginning with the disability case and looking at the many correlates of that disability (which are quite unclear and clouded), it might behoove us to address ourselves to the reading process and attempt to define some components that cut across all levels of reading ability. There ought to be a distribution of good and bad (to use value terms) in any of these components; perhaps on that basis, we could define more carefully the problem of the child who has difficulties in reading.

Most of us, first, are concerned with identification of an assumed process and, second, make the assumption that the process has something to do with reading. I was interested in Dr. Silver's results, for example, with perceptual training. I want to support the position that perhaps the 50 hr that were involved might have been better spent in training in a more directly related perceptual task–that is, reading–rather than in trying to train an underlying process that is presumed to have some effect on the reading task.

The child who has problems in learning to read often has associated, measurable visual–motor delays. My question is: Did the visual–motor delay cause the reading disorder, or did the failure to learn to read cause the visual–motor delay? That is, does the actual process of learning to read help a child to organize his own perceptual functions–horizontal, left–right, recognition of spatiotemporal associations, and so forth? That question has not been answered very carefully in the literature. What is the effect of learning to read on other aspects of perceptual organization?

That is the kind of question that I would hope a conference like this could get to: to attempt to define the component processes of reading, and then try to relate them to disability and success. Inasmuch as there are successful readers, I cannot believe that the processes that they use are very different from those used by the child with reading disability. The problem, then, is to define them critically and to see how they interact in achievers and nonachievers in reading.

I would like to comment on Dr. Hochberg's presentation, because I was interested in the use of paralinguistics. He pointed out that the youngster responds to inflection, gesture, and many other things in the auditory communication system. Yet, when we present a youngster with a reading task, we take away all those clues and present him with a page with only printed symbols on it; we remove gesture, inflection, and interpretation, so that he must now depend on visual perception alone, without all the other things that go along with oral communication. Thus, we are presenting him with a different kind of task.

DR. INGRAM: I found many of the conference papers difficult to understand. They were something of a challenge because of my lack of knowledge of experimental work in physiology, a great deal of which, of course, has gone on in this country in so many centers that it is almost impossible to keep up with the relevant literature. I think that the conference has considerable value for a mere clinician in indicating what advances are taking place.

Like the other speakers, I am very worried about the implications of the findings of mass surveys. If 20% or thereabout of children are unsuccessful to some degree in reading, as Eisenberg and many others have shown in this country and as has been shown in Great Britain, then this is a tremendous criticism, not of the children, but of our educational systems. Moreover, consider the figures from different types of schools. Eisenberg[2] found that 1% of the children were not doing well by a year and none by 2 years in his independent, expensive schools, whereas a very high proportion were doing better than expected. This may very well be due in part to social selection, but one cannot help wondering to what extent it depends on the size of the classes, the training of teachers, the early recognition of handicaps, and so on, when one compares what occurs in less favored schools.

In Glasgow, there are still some children receiving part-time education—such is the pressure on primary-school teachers. I know of several classes in which, after 2 years, the teachers do not even know the students' names. A similar situation has been described by Cazden[1] in this country. We have tended to concentrate, probably rightly, on the difficulties of individuals; only recently have we begun to consider the environmental difficulties to which they are exposed.

Perhaps we are overestimating the problem at the higher sociologic level of the relatively small numbers of children that come to clinicians, and overlooking the larger problem among disadvantaged children. About 250 or 300 children are referred to me each year because of educational problems. About one third of these, probably, I would classify as dyslexic or as having specific spelling or reading problems. A great many of the others are children of middle-class parents who want a "respectable" diagnosis of dyslexia, rather than an "unrespectable" diagnosis of mental retardation. I am trying to put things into focus and emphasize that we have been talking about a small minority of children.

We have a very interesting control group, a natural one in Edinburgh. A high percentage of children go to fee-paying middle-class schools, rather than state schools. These schools, by and large, although they have many male teachers in the primary grades, do not have any remedial-teaching system. In contrast, the state schools in Edinburgh have a highly developed remedial-teaching system, and about 8% of the children attend them at one time or another. Of the children who are referred to me because of possible learning difficulties, about 80% come from the middle-class fee-paying schools. That is partly because of parental drive, ambition, and anxiety about the educational attainments of their offspring, but I am sure that it is also due in part to the fact that remedial teaching at an early stage in the state schools is preventing a great deal of later disability. In fact, it is preventing the failure that results from failure.

I would very much like to be at another conference where the clinicians, the experimentalists, the brain scientists, and the ophthalmologists would be much more silent and let the talking be done by the teachers, those who organize education, and possibly some of the politicians who are so keen on education on election day and so neglectful between elections.

We have to ask to what extent poor reading is environmentally determined. If a child gets off to a bad start, if he begins to fail, then there is likely to be failure upon failure, and the vicious spiral with which we are all familiar. The child fails, and he is therefore given extra training (probably of the wrong type) and becomes progressively anxious; and, of course, in a state of anxiety, no one will achieve as well as he can. Children are referred to child-guidance clinics at the age of 10 who could probably have been spared this had they received just a little more consideration at the age of 5 or 6.

I am extremely concerned about the training of children in their areas of deficit. It is rather like the situation in cerebral palsy, when one ties the good hand of a child with hemiplegia to ensure the use of his bad hand. Particularly if sensation is lost in the bad hand, the child is more paralyzed than he was before. He becomes more afraid and disturbed, in addition to being basically handicapped. There is a tendency, in cerebral palsy, if the child does not respond to physiotherapy, to give more physiotherapy, instead of asking whether the physiotherapy is appropriate. I am afraid a great deal of remedial teaching today is based on a similar fallacy: When remedial teaching of a specific type does not appear to benefit these children, give more remedial teaching, rather than asking the rather simple question: "Is the basis for remedial teaching appropriate?"

DR. SHANKWEILER: I was struck by one characteristic of the visual system that was stressed by Dr. Sperling and Dr. Hochberg: its capacity for parallel processing. The eye can take in an enormous amount of information in only a tiny fraction of a second. If the eye is such an efficient channel, why is reading difficult for so many people? I think that Dr. Sperling has provided us with a valuable clue. The information taken in, in a single fixation, is useful to us for only a brief period, and the proportion of this information that can be used by the perceiver depends to a great extent on how quickly he can encode the information. Language is undoubtedly the most available and most ubiquitous code in human perception, and we have been shown how readily and how automatically observers encode visually perceived letter shapes, not into visual forms, but as speech (as letter names).

Being able to encode optical shapes as language requires knowledge of the rules that relate shape to sounds. Dr. Chall told us that some children need more explicit drill in these things than others in order to learn how to read, and I heartily second what Dr. Keogh and Dr. Robinson have said: We need to see a great deal more work done on the reading process and its development in normal children. The great bulk of research on reading has left many questions unanswered.

For example, we know that the rules that relate alphabetic symbols to language are more complex in some languages than they are in others. In some languages, such as Italian, the sounds of speech (the phonemes) map rather simply onto the alphabet. In other languages, such as English, this mapping is much more complex, and one must take into account levels of language other than the sound structure in order to generate the spelling rules. It ought to follow, then, that fewer reading disabilities should occur among Italian children than among English-speaking children. That constitutes a straightforward question, but I do not think we have the answer to it.

There is general agreement that many children do not read as well as they should, but I do not think we have made much progress in discovering why. If we really want to find out, we have to ask some specific questions about the characteristics of language and perception in children who can and cannot read. We all agree, I suppose, that a requirement for learning to read is rapid and accurate identification of the letter shapes. How many children who cannot read fail at this level? That is another straightforward question, but, again, I think that we do not know the answer. For children who can pass this test, we can then inquire whether they have learned the rules that relate letter shapes to sounds. That is a large task. It requires a detailed examination of the kinds of errors that children make in reading and an attempt to relate them to aspects of the acquisition of spoken language. Dr. Ingram's work shows that we will surely find these relationships, but we know almost nothing about them as yet. Some of us at Haskins Laboratories and the University of Connecticut find this problem challenging.

The point I want to make is that there are a number of important questions about learning to read that could be answered but that usually have not been asked in research on reading and reading disability. The answers would provide the skeleton of a classification system that would permit the sorting of children who cannot read into scientifically useful categories. As long as we lack an empirically based system of classification, no rational approach to treatment is possible.

DR. ROBINSON: I think we have to know, as Dr. Keogh has pointed out, whether a deficit must be corrected to some degree to promote adequate reading. I am concerned, for example, about those who approach the teaching of reading from an auditory point of view, as though it were never necessary for the child to perceive visually. It seems strange that we can talk about teaching by using strengths and neglecting deficits, if those deficits involve absolutely essential parts of the totality of learning. I want to comment on this primarily so that none of us will leave this conference believing that he can capitalize on the strengths and neglect essential deficits. For example, visual perception is essential because reading is a visual task. How to diagnose it and how to correct it are the major problems, and not whether it is necessary. Would you agree, Dr. Ingram?

DR. INGRAM. I agree entirely with that. Obviously, some basic skills are necessary. But if a child fails, for example, in recognizing word shapes, then I would not spend my time teaching him by "look and say." I think I would encourage him to use the phonic approach, but I agree that he must first reach the stage of learning where he is able to read letters.

DR. ROBINSON: Over and over again, the question has been raised about more boys than girls having reading disabilities, and we find this consistently. A study that I don't believe has been mentioned, but is of interest, was done by Ralph Preston[6] with a colleague in Germany. It began as a comparative study of the achievement of German and American children, but one of the surprising findings was that in Germany more girls than boys had difficulty in reading. This leaves us with a question of considerable interest to explain.

DR. INGRAM: In a classic study, Hallgren[3] found that many more boys than girls were referred to reading clinics, but that the incidence in boys and girls of what he called "specific dyslexia," which he defined in his own terms, was approximately similar. I was reminded of this during Dr. Kagan's presentation, when he mentioned the differences in behavior in boys and girls. This is the sort of difference that leads immediately to a research project. In particular, Hallgren found, when he studied the families of the children referred to him, that the girls were also affected, but not so severely. Therefore, we are left with the simple question: "What is it that makes boys more dyslexic and makes them have more difficulties than girls?" Obviously, we are dealing with a multifactorial situation. I have mentioned environmental factors already. Here is an intrinsic factor, and there must be hundreds of other factors in early upbringing that we do not know about but that determine whether a child will suffer from significant reading difficulty.

DR. MEIER: Dr. Kagan, I, and others have touched on the male-teacher and male-classroom possibilities. Our experiments with kindergarten and first-grade children in Greeley indicate that the all-male groups do as well as the all-female and better than the mixed groups in reading achievement. I wonder whether Dr. Robinson was getting at this when she said that in Germany the sex ratio of reading problems was reversed, because they use male teachers for young children more than we do.

Frances McGlannan[4] is doing a study dealing with genetic anomalies in poor readers. She believes that her evidence indicates that there is a higher incidence of sex-linked genetically determined problems with poor readers. We are doing a gross chromosomal analysis, the buccal smear test, to see whether there are any indications of sex-linked anomalies in children with learning disabilities. If there are, we plan to do a total chromosomal analysis on the child. I submit that this problem is susceptible to research. The answers are elusive and complex.

Dr. Sperry has indicated that boys seem to have less-stable spatial predictability, in terms of organization mediated by the right hemisphere, than do girls. Dr. Kagan suggests that the boys merely behaved differently and therefore responded differently. I would suggest that there is some interaction between the brain and the behavior, if you will, and a corresponding interaction between those and the teacher in the classroom.

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DISCUSSION

DR. ROBINSON: An international study on achievement in mathematics has already been published. (Husén, T., Ed. International Study of Achievement in Mathematics: a Comparison in 12 Countries. 2 vol. New York: John Wiley & Sons, 1967. 304 and 368 pp.) I was reminded of it when the question of dyslexia in Japan came up, because it was found that arithmetic and mathematical achievement of Japanese children at various age levels was ahead of that in any of the other countries studied. Having completed that study, the international group is now assessing achievement at different age levels in various other content areas and in reading. The committee is developing an achievement test that will be as fair as possible in reading and in writing for children in various countries. I think we need to look to that (it is already in progress) to make some comparisons between the kinds of achievements and disabilities that are found in countries with various language characteristics.

DR. DENENBERG: I came to this conference with the belief that dyslexia was a specific human syndrome. That is definitely wrong. First, there are many correlative events: visuomotor coordination, spatial disturbances, perceptual problems, and pattern-recognition problems; and also there happen to be reading difficulties. The culture in which we live is such that we have focused on reading difficulties. But we have made them central rather than merely correlative. What impresses me is that all the problems except reading occur in higher mammals. I therefore submit that, although we are focusing on reading disabilities, they are merely a symptom of a much more basic problem, which is probably not unique to humans but runs through a wide variety of higher mammals. And the way to approach the problem from a research point of view is to look at it both at a human level and at a lower-primate level.

DR. RICHARDSON: I should like to add a caution to whatever is done experimentally in nonhuman primates. Reading is a language function, as we have spent some time discussing here. We must not lose sight of that facet in extrapolating experimental observations on nonhuman primates. The problem goes beyond the perceptual aspects. I think the visual aspects are important, but I believe we have ample evidence from research that auditory discrimination, sequencing, and language development are as important as visual perception. I referred to visual perception earlier only to point out that it was one of a number of factors.

DR. GUNDERSON: I would like to comment on the definition and incidence of dyslexia and reading disabilities. The Interdisciplinary Committee on Reading Problems is composed of professionals representing many disciplines—psychology, education, sociology, linguistics, anthropology, economics, child development, and specialized fields of medicine, such as ophthalmology, pediatrics, neurology, and psychiatry. One of the problems faced by the Committee was terminology: Although workers in various disciplines used the same terms, discussion revealed that the meanings of terms varied according to the discipline of the user. The Committee members were concerned with reading problems, but it was difficult to obtain a consensus on what a reading problem was or to define reading disability. It was apparent that common terminology was essential for effective communication. So the group agreed to compile a glossary (Gunderson, D. V. Reading problems: glossary of terminology. Reading Research Quarterly 4:534–547, 1969) to use during the writing of the several manuscripts that will be published in a single volume by the Center for Applied Linguistics and to appear as an appendix to the volume. One of the seven Committee task forces is concerned with incidence and implications. We have found, as most of us already knew, that in the United States we have no accurate estimate of the number of children who are disabled in reading; we have only an educated guess. The primary reason for the lack of an estimate is that reading disability itself is defined in many different ways. Most workers in education would feel that an estimate of 15%–20% of children as dyslexic is rather high.

DR. YOUNG: But would it be high in terms of reading disability if that is defined in terms of "years retarded"?

DR. GUNDERSON: We still have to define "reading disability" and "dyslexia." Many children who are a year retarded in reading are retarded simply because of poor educational experiences and have no physical or neurologic problems.

DR. BOYNTON: I have been struck by the lack of mention of an active area of research and a related point of view. Although I am not expert in it, I think this approach ought at least to be mentioned and put on the record. I refer to the use of programmed instruction or teaching machines. I know that active research is going on with such things as typewriters connected to computers that are operated by very young children. I submit that, although the concept of training children in how to read through the use of apparatus that requires a computer may seem way out today, this sort of approach might seem quite feasible in the near future. One of the nice things about computers is that they are precise and have infinite patience. Another good thing about computers is this: to write a program, one needs an extremely precise idea of what he wants the machine to do for him; therefore, he must sharpen his thinking about the process involved. In the pattern of the educational system that we are generally using in this country today, material is given to 30 or 40 students. Some succeed and some do not: that leads to failure on the part of those at the bottom of the group and the beginning of the downward spiral referred to earlier.

I think that Professor Skinner at Harvard, who has been one of the motivating forces behind the concept of programmed learning, and many others have pointed out that, through the use of programmed instruction, an entirely different approach can be taken. Rather than throwing a given quantity of material at the students and seeing whether they can handle it, we can allow each student to proceed at his own pace and arrange things, it is hoped, so that success occurs and failure is almost ruled out.

DR. YOUNG: About 5 years ago, we started a group of very deprived 4-year-olds on programmed typewriters. These children are now in school and performing very well in reading. The prognosis would have been that at least one third of them would have been placed in mental-retardation classes; not one of them has been placed there. They are exceeding the control group, and even doubling their scores in several achievement tests. We intend to follow them for 12 years. Your point about the rigor involved in programming and the student's control over the pace at which the material is presented is extremely important. I would also like to say that, even if you do not have a computer or automated equipment for presenting data, teachers can be trained to function in a facilitative fashion, rather than in a turkey-stuffing fashion—jamming data down the unwilling throats of children. The former style of instruction is considerably more effective. Your points are germane to one of the sore spots in education.

DR. ULLMANN: I would like to refocus attention on a point that Dr. Keogh made earlier: the question of continuity versus discontinuity in the process we call reading.

I am aware that, from the standpoint of science, it may be proper to consider the components of the reading process a monotonic system and yet call the disability a definably different, qualitatively separate phenomenon. This seems to be so; in the children to whom Dr. Robinson has referred, reading was improved in a significantly large proportion by improving the reading instruction. I think there was a reduction in reading disability from 20% to 5%. But the 5% remain, so that one would assume that the improvement in instruction had made its contribution, which could continue, but that there might be something within the learner that also has to be considered, in addition to the instructional system.

Similarly, there is a situation with which I have had some familiarity in the last few years: the effort by the armed forces to accept 100,000 men per year who would previously have been rejected, through improvement in instructional material and adjustment of training procedures. This is of benefit not only to the armed forces, but also to the men themselves.

The person who has what we might loosely call dyslexia remains in a category separate from those who can be reached by improvements in methods for general use. Most people may have been effectively instructed, but there still seems to be a small proportion who cannot be dealt with easily within a mass approach. I do not mean that as a social policy this concern with methods of general applicability is unwise; but I want to highlight the fact that we still seem to have a problem with residual disability. I would ask whether the concept of discontinuity must be faced if we are to reach the 100% of the children that Dr. Richardson alluded to in the further development of our educational and health system.

MRS. GAARDER: I would like to ask the panel members whether they can look into their crystal ball and foresee not only a speech therapist to screen children (which is done now), but a hearing therapist and a reading therapist. Perhaps after Dr. Shankweiler works out his classification of reading problems, these specialists may be able to identify children in need of special training before failure begins and to serve a very important purpose.

DR. ROBINSON: That has been my goal for 20 years. Following this conference, however, I am beginning to wonder whether children should be screened before they even enter school. It seems to me that the screening process ought to come much earlier than 6 years of age, when children enter school in this country, or 5, when they enter kindergarten. At this time, I do not know what to screen or how to screen. This precision is the goal that we simply must achieve, because prevention is our ultimate goal.

Participants and Other Contributors

RICHARD B. ADAMS, c/o Adams Associates, 50 Simpson Court, Wallingford, Connecticut 06492

MATHEW ALPERN, Ph.D., Professor of Physiological Optics, Departments of Ophthalmology and Physiology, University of Michigan Medical Center, Ann Arbor, Michigan 48104

WILLIAM R. BALDWIN, O.D., Ph.D., Dean, College of Optometry, Pacific University, Forest Grove, Oregon 97116

EDGAR A. BERING, JR., M.D., Special Assistant to the Director, National Institute of Neurological Diseases and Stroke, Bethesda, Maryland 20014

ROBERT M. BOYNTON, Ph.D., Director, Center for Visual Science, University of Rochester, Rochester, New York 14627

PIERRE BUSER, Professeur, Laboratoire de Neurophysiologie Comparée, Faculté des Sciences, Université de Paris, Paris 5^{e}, France

JEANNE S. CHALL, Professor of Education, Graduate School of Education, Harvard University, Cambridge, Massachusetts 02138

ALEXANDER L. CLARK, Ph.D., Staff Associate, Committee on Life Sciences and Social Policy, National Research Council, Washington, D.C. 20418

DAVID A. COGAN, M.D., Director, Howe Laboratory of Ophthalmology, Massachusetts Eye and Ear Infirmary, Boston, Massachusetts 02114

VICTOR H. DENENBERG, Ph.D., Professor of Biobehavioral Sciences, University of Connecticut, Storrs, Connecticut 06268

ROBERT W. DOTY, Ph.D., Professor of Physiology, Center for Brain Research, University of Rochester, Rochester, New York 14627

ROBERT L. FANTZ, Ph.D., Professor of Psychology, Case Western Reserve University, Cleveland, Ohio 44106

RICHARD FEINBERG, Ph.D., Program Analyst for Vision and Diseases of the Eye, National Institute of Neurological Diseases and Stroke, Bethesda, Maryland 20014

MERTON C. FLOM, O.D., Ph.D., Professor of Physiological Optics and Optometry, College of Optometry, University of California, Berkeley, California 94720

KENNETH R. GAARDER, M.D., Staff Psychiatrist, Veterans' Administration Hospital, Washington, D.C. 20422

MARIE S. GAARDER, Speech Therapist, St. Maurice School for Retarded Children, Bethesda, Maryland 20014

MITCHELL GLICKSTEIN, Ph.D., Associate Professor of Psychology, Brown University, Providence, Rhode Island 02912

DORIS V. GUNDERSON, Ph.D., Executive Director, Interdisciplinary Committee on Reading Problems, Center for Applied Linguistics, Washington, D.C. 20036

ROSA A. HAGIN, Ph.D., Department of Neurology and Psychiatry, New York University Medical Center, New York, New York 10021

IRA J. HIRSH, Ph.D., Director of Research, Central Institute for the Deaf, St. Louis, Missouri 63110

JULIAN HOCHBERG, Ph.D., Professor of Psychology, New York University, New York, New York 10003

THOMAS T. S. INGRAM, M.D., F.R.C.P.E., D.C.H., Senior Lecturer in Paediatric Neurology, Department of Child Life and Health, University of Edinburgh, Edinburgh 9, Scotland

RICHARD JUNG, Dr. med., Professor of Neurology and Neurophysiology, Universität Freiburg, 78 Freiburg i. Br., Germany

JEROME KAGAN, Ph.D., Professor, Department of Social Relations, Harvard University, Cambridge, Massachusetts 02138

JAMES F. KAVANAGH, Ph.D., Health Scientist Administrator for Human Communication, National Institute of Child Health and Human Development, Bethesda, Maryland 20014

BARBARA KEOGH, Ph.D., Assistant Professor, Area of Special Education, School of Education, University of California, Los Angeles, California 90024

LORRAINE LANGE, Ph.D., Professor of Education, State University College, Buffalo, New York 14222

WILLIAM LIGHTFOOT, Ph.D., UNESCO Adviser to UNICEF, United Nations, New York, New York 10017

DONALD B. LINDSLEY, Ph.D., Professor of Psychology and Physiology, Brain Research Institute, University of California, Los Angeles, California 90024

LEWIS P. LIPSITT, Ph.D., Director, Child Study Center, Department of Psychology, Brown University, Providence, Rhode Island 02912

PHILLIP LISS, Ph.D., Department of Psychology, City College, City University of New York, New York 10036

WILLIAM M. LUDLAM, O.D., Director, Research Laboratory, Optometric Center of New York, New York, New York 10036

ELWIN MARG, Ph.D., Professor of Optometry and Physiological Optics, School of Optometry, University of California, Berkeley, California 94720

LOUISE H. MARSHALL, Ph.D., Professional Associate, Division of Medical Sciences, National Research Council, Washington, D.C. 20418

WADE H. MARSHALL, Ph.D., Chief, Laboratory of Neurophysiology, National Institute of Mental Health, Bethesda, Maryland 20014

MARY WOOTTON MASLAND, Research Associate, Columbia University School of Medicine, New York, New York 10032

RICHARD L. MASLAND, M.D., Chief, Department of Neurology, College of Physicians and Surgeons, Columbia University, New York, New York 10032

WILLIAM A. MASON, Ph.D., Head, Behavioral Sciences, Delta Regional Primate Research Center, Covington, Louisiana 70433

JOHN H. MEIER, Ph.D., Associate for Program Management, Individual Learning Disabilities, Rocky Mountain Educational Laboratory, Greeley, Colorado 80631

WILFRID RALL, Ph.D., Mathematical Research Branch, National Institute of Arthritis and Metabolic Diseases, Bethesda, Maryland 20014

R. J. REICHLER, M.D., Extramural Research Division, National Institute of Mental Health, Chevy Chase, Maryland 20016

H. BURTT RICHARDSON, JR., M.D., Assistant Professor of Pediatrics, George Washington University School of Medicine; and Research Associate, Department of Pediatric Neurology, and Attending Pediatrician, Children's Hospital of the District of Columbia, Washington, D.C. 20009

AUSTIN H. RIESEN, Ph.D., Professor of Psychology, University of California, Riverside, California 92502

HELEN M. ROBINSON, Research Professor of Reading, Emeritus, Department of Education, University of Chicago, Chicago, Illinois 60637

A. RUIZ-MARCOS Ph.D., Sección de Neuroanatomía Comparada, Instituto Cajal, Velazquez 144, Madrid 6, Spain

DELWYN C. SCHUBERT, Ph.D., Reading Clinic Director, Department of Education, California State College, Los Angeles, California 90032

DONALD SHANKWEILER, Ph.D., Haskins Laboratories, 270 Crown Street, New Haven, Connecticut 06510

ARCHIE A. SILVER, M.D., Department of Neurology and Psychiatry, New York University Medical Center, New York, New York 10021

GEORGE SPERLING, Ph.D., Bell Telephone Laboratories, Murray Hill, New Jersey 07971

ROGER W. SPERRY, Ph.D., Professor of Psychobiology, Division of Biology, California Institute of Technology, Pasadena, California 91109

LOTHAR A. SPILLMANN, Ph.D., Department of Retina Research, Retina Foundation, 20 Staniford Street, Boston, Massachusetts 02114

RALPH C. STAIGER, Ph.D., Executive Secretary, International Reading Association, P.O. Box 119, Newark, Delaware 19711

CHARLES A. ULLMANN, Ed.D., Executive Director, Secretary's (HEW) National Advisory Committee on Dyslexia and Related Reading Disabilities, National Institutes of Health, Bethesda, Maryland 20014

KLAUS R. UNNA, M.D., Chairman, Department of Pharmacology, University of Illinois College of Medicine, Chicago, Illinois 60680

F. VALVERDE, M.D., Jefe de Sección de Neuroanatomía Comparada, Instituto Cajal, Velazquez 144, Madrid 6, Spain

MILTON A. WHITCOMB, Ph.D., Executive Secretary, Committee on Vision, National Research Council, Washington, D.C. 20418

JERRY B. WURSTER, M.D., Howe Laboratory of Ophthalmology, Massachusetts Eye and Ear Infirmary, Boston, Massachusetts 02114

FRANCIS A. YOUNG, Ph.D., Director, Primate Research Center, Washington State University, Pullman, Washington 99163

Glossary

The jargon and technical terms of one discipline are deterrents to understanding by readers trained in another discipline. This glossary is intended to relieve the irritation of reading material in an unfamiliar subject and to promote comprehension. It consists of terms that are not explained by the speakers in context; the definitions are confined to the sense in which they are used. Terms in italics are defined elsewhere in the glossary. Specialists in the several disciplines have been consulted, as have the following authoritative sources:

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ACCOMMODATION: A change in thickness, curvature, and power of the lens of the eye through action of the *ciliary muscle* to adjust the eye for near vision. Accommodation is usually accompanied by changes in pupillary diameter and *convergence* of the eyes.

"ACUTE" INVESTIGATIONS: In neurophysiology, usually investigations on anesthetized animals that are sacrificed at the termination of the experiment, in contrast to "chronic" animals, which are used more than once.

AMACRINE CELLS: Intraretinal association neurons without long extensions, acting on synaptic junctions between *bipolar* and *ganglion cells*; one of several types of association cells facilitating or inhibiting retinal neural activity. See also *horizontal cells*.

AMBLYOPIA: Reduction in visual acuity that cannot be improved with lenses or attributed to disease.

AMETROPIA: An abnormality of the eye, such that when it is at rest, light rays are not focused on the retina, but rather in front of or behind it. Includes *myopia, hyperopia*, and *astigmatism*.

ANTERIOR COMMISSURE: One of the transverse bands of nerve fibers that connect the two cerebral hemispheres.

APHASIA: Impairment of the ability to use language because of defect or impairment of the brain, not sensory impairment of vision or hearing.

APRAXIA: Inability to carry out purposeful movements in the absence of identifiable paralysis or other motor or sensory impairment.

AREA 17: See *striate area*.

AREA 18: An area of the *occipital lobe* of the cerebral cortex, mainly on the dorsal surface of the lateral gyrus adjacent to *area 17*, showing electrical activity after stimulation of the visual tract. Also called "visual II" and "secondary visual cortical area."

ASTIGMATISM: Lack of symmetry of curvature of the refractive surfaces of the eye as a result of which a ray of light is not focused on the retina sharply in all meridians.

ATAXIA: Failure of muscular coordination.

ATROPINE: A chemical compound found in plants that, among other uses, dilates the pupil and paralyzes the *ciliary muscle* controlling accommodation of the lens when applied locally to the eye.

AUDIOGRAM: A plot of the acuteness of a subject's hearing at various sound frequencies.

AUDITORY BLENDING: The ability (1) to make sense out of the parts (*phonemes*) of a word pronounced with separations between phonemes and (2) to obtain an approximation of the intended word sufficient for recognition of the word.

A-WAVE: Negative deflection of the *electroretinogram* occurring in the light-adapted eye following stimulation by a flash of light, or in the dark-adapted eye by a red light, and considered to be related to *cone* activity.

AXON: A nerve-cell expansion or process that is typically single and long, terminates in short branches relatively far from the cell body, and usually conducts impulses away from the cell body.

AXOSPINODENDRITIC CONTACTS: *Synapses* between *dendritic spines* of one neuron and *axons* of another neuron.

BENDER-GESTALT TEST: A performance test requiring reproduction of the configuration in line drawings.

BENTON VISUAL RETENTION TEST: A test of visual perception that depends on *visual-motor* ability.

BIPOLAR CELLS: Two types of connecting cells in the retina: monosynaptic bipolar cells, which connect *cones* to nerve fibers and may permit direct transmission to the brain; and polysynaptic bipolar cells, which connect both *rods* and *cones* and nerve fibers and permit interactions between *receptors* before transmission to the brain.

BUCCAL SMEAR TEST: Microscopic examination of cells scraped from the inside of the cheek and stained to determine chromosomal irregularities.

B-WAVE: Positive deflection of the *electroretinogram* following stimulation by a flash of light; largest in poor illumination and with blue light. Thought to be produced mostly by *rod* activity.

CEREBRAL AQUEDUCT: A narrow canal about 3/4 in. long extending from the third ventricle to the fourth ventricle in the midbrain region.

CEREBRAL COMMISSURES: Neural tracts connecting the two sides of the cerebrum. Examples are the *corpus callosum* and *anterior commissure*.

CEREBRAL DOMINANCE: A functional asymmetry in which one side of the brain assumes greater control of cerebral functions than the other side, demonstrated by *laterality* in voluntary motor acts. See also *laterality* and *dominant laterality*.

CILIARY MUSCLE: The smooth (involuntary) muscle that affects the shape of the lens in visual *accommodation*.

CLOSED COLONY: An inbred colony.

CODE-EMPHASIS PROGRAMS: Programs of teaching reading that emphasize sound-symbol relationships. See also *meaning-emphasis programs*.

COMMISSUROTOMY: Surgical sectioning of the connecting bands of a *cerebral commissure*. In the brain, surgical separation of the neural connections between the two hemispheres.

CONE: A photoreceptor neuron characterized in part by being thicker than a *rod*, by the complexity of its *bipolar* connections, and by use chiefly in day vision;

occurs most densely in the *fovea centralis* area of the retina, where visual acuity is highest.

CONSENSUAL PUPILLARY RESPONSE: Similar reaction of both pupils to a stimulus applied to only one eye.

CONVERGENCE: In binocular vision, the coordinated movement of the two eyes toward *fixation* on the same near point. Neural convergence is the *synapsing* of many cells with fewer cells at successively lower or higher levels.

CORPUS CALLOSUM: A large bundle of transverse nerve fibers connecting the cerebral hemispheres.

COUNTERBALANCING: Technique used in experimental design for controlling continuation effects by presenting stimuli in balanced sequences (such as AB, BA).

CRITERION: In experimental psychology, a predetermined level of performance usually less than a perfect score.

d-c POTENTIALS: The so-called steady or slowly changing electrical potentials that can be observed in various parts of the brain under constant observation conditions.

DECIBEL (db): Nontechnically, a unit for measuring the relative loudness of sounds approximately equal to the smallest degree of difference of loudness ordinarily detectable by the human ear, whose range includes about 130 db on a scale beginning with 1 for the faintest audible sound.

DECUSSATION: A crossing over in the visual system of optic fibers at the *optic chiasma*.

DEEP STRUCTURE: Elements of a sentence that determine its semantic interpretation, including grammatical relations, functions, and categories.

DENDRITE: A protoplasmic expansion or process of a neuron that usually terminates in a highly branched state relatively near the cell body and conducts impulses toward the cell body. Dendrites of pyramidal cells are termed apical dendrites or basilar dendrites depending on where they leave the cell body, which is oriented with the apex toward the cerebrocortical surface.

DENDRITIC SPINE: A short bulbous expansion of the *dendrite*, with which the *axon* endings from another neuron make contact; spines are distributed along the dendrite according to distance from the cell body and other factors.

DETOUR BEHAVIOR: A behavior pattern that seeks to reach a goal by an indirect route in order to avoid an unpleasant or apparently impassable barrier.

DIOPTER: A unit of measurement of the refractive power of a lens; equal to the reciprocal of the focal length in meters.

DIPLOPIA: Double vision—two simultaneous visual images of a single object.

"DISABLED READER": See *reading disability*.

DISHABITUATION: Restoration of a response that has weakened because of repeated presentation of the stimulus.

DISINHIBITION: Lessening the reciprocal inhibition caused by excitation of a neuron by means of excitation of another neuron.

DOMINANT LATERALITY: The preferential use in voluntary motor acts of ipsilateral members of the different pairs of organs, as the right ear, eye, hand, leg (dextrality) or the left ear, eye, hand, leg (sinistrality). There is a general tendency to be consistently right-dominant or left-dominant, but mixed and crossed dominance are also found.

DYSLEXIA: From "dys"–faulty, impaired–and "lexis"–speech, from *legein* (Greek) to speak; confused with *legere* (Latin) to read, the medically more common interpretation, as in alexia. See *reading disability*.

DYSPRAXIA: Partial loss of ability to perform coordinated movements; nonspecific clumsiness; a sign considered indicative of possible neurologic involvement.

EDGE-DETECTOR UNITS: Retinal *rods* or *cones* that respond to contrasting luminance boundaries such as that provided by the border between black and white areas.

EDGE GRADIENT: The gradual variation in illumination at an edge between a bright field and a less bright field due to the *optical-spread function* and eye micromovements.

ELECTROANATOMIC STUDIES: Deduction of probable anatomic pathways from the pattern of nerve impulses recorded at different sites.

ELECTRORETINOGRAM (ERG): Record of changes in electrical potential in the retina after stimulation by light; may be obtained in man by placing one electrode on an anesthetized cornea and the indifferent electrode in the mouth.

EMMETROPIA: A state of proper correlation between the refractive system of the eye and the axial length of the eyeball such that rays of light entering the eye parallel to the optic axis are brought to a focus on the retina.

ENUCLEATION: Removal of the eyeball after the *extraocular muscles* and *optic nerve* have been severed.

EVOKED POTENTIALS: Electrical signs of neuronal activity resulting from stimulation of nerves or sensory end-organs.

EXTINCTION: Reduction or elimination of a conditioned response by not reinforcing the response.

EXTRAOCULAR MUSCLES: Three pairs of muscles (the superior and inferior recti, the internal and external recti, and the superior and inferior oblique muscles), arranged around the eyeball that act in a coordinated manner to rotate the eyeball and maintain appropriate *convergence* with the other eyeball.

FACIAL DIPLEGIA: Paralysis of both sides of the face.

FEATURE EXTRACTION: The process of separating from the total input the particular characteristics of a sensory stimulus that are recognized.

FIXATION: The direction of gaze is such that the image of an object looked at falls on the *fovea centralis*.

FOOT LAMBERT (fL): A unit of *luminance* equal to the luminance of a perfectly diffusing surface that emits or reflects one lumen per square foot. A lumen equals the light emitted (in a unit solid angle) by a uniform point source of one candle intensity.

FORMANT: The frequencies (or groups of frequencies) that characterize the timbre of a sound and distinguish it from sounds of different timbre.

FOVEA CENTRALIS: A tiny depression, about 1 deg wide in man, in the center of the *macula lutea retinae*, where the layers of the retina are spread apart, permitting light to fall directly on the *cones*; the part of the retina that provides the best visual acuity and most sensitive *visual discrimination*.

FOVEAL EXAMINATION: Examining an object with the most discriminative part of the retina, the *fovea centralis*; involves coordinated turning of the eyes so the optic axes converge on the *fixation* point.

FUSIONAL MOVEMENTS: Movements of the eyes that bring the retinal images of the same part of a visual stimulus sufficiently close to corresponding points of the retina that a single image is perceived.

GANGLION CELLS: In the retina, neurons representing the third link in the chain that carries visual stimuli to the brain, whose cell bodies and *dendrites* are in the retina and whose *axons* constitute the fibers of the *optic nerve.* Retinal ganglion cells differ greatly from one another in body size, location, and manner in which they make synaptic connections; they are activated by *bipolar, horizontal*, and *amacrine cells*.

GATING FUNCTION: Selection on an arbitrary basis—for example, voltage of neuron spikes—in recording techniques; also applies to all kinds of neurophysiologic phenomena that can be segregated.

GENERALIZATION: In psychology, the ability of an associated or similar stimulus to cause a response produced by a particular stimulus, especially when of the same sense mode.

GESTALT: A structure or configuration including shape, pattern, and time, of physical, biologic, or psychologic phenomena so integrated as to constitute a functional unit with properties not derivable from its parts in summation.

GESTALT PSYCHOLOGY: The study of perception and behavior from the standpoint of an organism's response to configurational wholes, with stress on the identity of psychologic and physiologic events.

GOLGI-COX PREPARATIONS: Preparation and staining of nerve tissues for histologic examination by the Cox modification of the first neurohistologic method, devised by Camillo Golgi 100 years ago. The method consists of staining suitably fixed and hardened slices of tissue with a weak silver nitrate solution; only nerve cells are blackened. The Cox modification results in improved staining of *dendrites.*

GRAPHEME (syn. letter, phonogram): A functionally distinctive graphic symbol, used in the writing system of a particular language.

HABITUATION: The waning of a response as a result of repeated stimulation that is not followed by reinforcement; distinct from fatigue and sensory adaptation.

HAPTIC SENSATIONS: Sensations arising from the touch *receptors* in the skin.

HEMIPLEGIA: Paralysis of one side of the body.

HERTZ (Hz): A unit of frequency of a periodic process equal to one cycle per second; replaces cycles per second (cps).

HORIZONTAL CELLS: In the retina, one of the types of association cells whose function is to distribute photogenic impulses in the retinal tissue itself and by facilitation, inhibition, or some similar influence to modify the impulses transmitted to the cerebrum. The horizontal cells interconnect *cone receptors* with *cones* or *rods* and are so named because of the horizontal spreading of their axons. See also *amacrine cells*.

HYPEROPIA (syn. hypermetropia, farsightedness): An optical abnormality of the eye in which, when it is at rest, rays of light entering the eye parallel to the optic axis are brought to a focus behind the retina.

INITIAL TEACHING ALPHABET (ITA): A notation system using the conventional symbols of English plus some others designed to represent particular sounds that are variably represented in English.

INNER PLEXIFORM LAYER: The relatively cell-free layer of the retina between the ganglion layer and the inner nuclear layer; consists mainly of *synapses*.

INTRACORTICAL ASSOCIATION CELLS: Neurons carrying out an associative function between cortical neurons.

KINESTHESIA: A sense mediated by *receptors* that lie in the muscles, tendons, and joints and are stimulated by bodily movements and tensions.

LABYRINTH: See *vestibular apparatus*.

LATENCY: Time between application of a stimulus and the resulting response at some point in the neural system.

LATERAL DOMINANCE: See *dominant laterality*.

LATERAL GENICULATE NUCLEUS: A group of neuronal cell bodies arranged in six layers. Nerve fibers from the retina of the contralateral side are received in layers 1, 4, and 6, and from equivalent spots of the ipsilateral retina, in layers 2, 3, and 5.

LATERALITY: Awareness of the two sides of one's body and ability to identify each correctly as left or right.

LATERAL SPECIALIZATION: See *dominant laterality*.

LAYER IV: The fourth layer from the surface of the primate *visual cortex* characterized by relatively small, densely spaced cells, including *stellate cells*.

LAYER V: The fifth layer from the surface of the primate *visual cortex*; characterized by large cells.

LIGHT TRAP: A device that eliminates unwanted light to produce an area of uniform illumination.

LONG-TERM MEMORY: Retention of information for longer than about a minute; usually, long-term memory refers to days, months, or years.

LUMINANCE: Refers to the effectiveness of a given light intensity on the visual system. Formerly called "brightness," and frequently confused with the brightness or physical energy of a light source itself. See also *Mach bands*.

MACH BANDS: Bright and dark rings noticed by Ernst Mach. When rapidly rotating disks of colored or black and white sectors are viewed, the successive sectors appear to fuse into a single color and intensity; the greater proportion of white to black (or one color to another), the brighter (or more like the better presented color) the rotating disk appears. When the proportions are not uniform, Mach bands appear. The explanation of the brightness-contrast effect is based on the mutual dependence of neighboring retinal points due to the retinal neural network.

MACROELECTRODE: A relatively large electrode (up to 0.5 mm in diameter) with low electrical resistance, used to record potentials accompanying reactions of aggregations of neurons.

MACULA LUTEA RETINAE: The part of the retina distinguished from the remainder of the retina by its yellow appearance; in the center lies the *fovea centralis*, the area of greatest visual acuity.

MEANING-EMPHASIS PROGRAMS: Programs of teaching reading that emphasize language meaning, as distinct from *code-emphasis programs*.

MESENCEPHALON (syn. midbrain): The middle division of the brain lying between the forebrain and hindbrain.

METHYLENE BLUE PREPARATIONS: Tissues prepared for study by a method devised by Paul Ehrlich (1886) consisting of injection of a weak solution of methylene blue into a living animal, whose nerve tissue decolorizes the blue dye. When the tissue is exposed to air, the nerve structures appear selectively stained in blue.

MICROELECTRODE: An electrode 1–3 μ in diameter used for intracellular recording of potentials accompanying reactions of single units.

MÖBIUS SYNDROME: Voluntary abstinence from motion owing to intense pain accompanying muscular movements.

MOLAR BEHAVIOR: A unit of behavior, described in nonphysiologic, psychologic constructs and made up of smaller specific units elicited by specific stimuli.

MOLECULAR BEHAVIOR: A small unit of behavior generally defined in terms of specific movements or of specific movements elicited by specific stimuli, or in physiologic terms.

MYELINATION: The condition of some nerve fibers that are covered with a fat-like substance (myelin) as a sheath.

MYOPIA (syn. nearsightedness): An optical abnormality of the eye in which, when it is at rest, light rays parallel to the optic axis are focused in front of the retina, rather than on it.

NEURAL GRADIENT: Variation in response of a neural system over a space distribution.

NODAL POINT: One of two points on the axis of an optical system so situated that a ray falling on one will produce a parallel ray emerging through the other.

NOISE: Interference of variable intensity that forms a diffuse background against which a specific stimulus effect occurs. Within neural systems, "noise" is the residual on-going activity, and any signal to be effective must be higher in intensity than this background noise. See also *visual noise*.

NUCLEUS (pl. nuclei): In neuroanatomy, a general term used to designate a group of nerve-cell bodies usually within the central nervous system and bearing a direct relationship to the fibers of a particular nerve.

OCCIPITAL LOBE: The posterior lobe of the cerebral hemisphere, merging laterally with the *temporal lobe*, and concerned primarily with visual input.

OCULAR ASTHENOPIA: Weakness or rapid tiring of the eyes attended by pain in the eyes, headache, and dimness of vision.

OCULOMOTOR (syn. ocular-motor): Pertaining to the movements of the eye.

OCULOMOTOR APRAXIA: Loss of ability to move the eyes at will or to follow a moving object to either side.

OCULOMOTOR COORDINATION: Reciprocal innervation of the pairs of *extraocular muscles*, resulting in coordination of movement of the eyeball on a point of fixation.

OCULOMOTOR DYSLEXIA: Reading disability due to oculomotor dysfunction.

OIL-IMMERSION PHOTOMICROGRAPH: A photomicrograph taken through a high-power lens having a clear oil phase instead of air between the lens and the object (or cover glass). The magnification is usually about 2,000 times, depending on the power of the objective and ocular lenses.

OMMATIDIUM (pl. ommatidia): An elongated unit of a compound eye of an arthropod, consisting typically of an external corneal lens beneath which is a crystalline cone and below it a rod-shaped form enclosed in a sensitive retinal element protected by pigment.

OPERANT CONDITIONING (syn. instrumental conditioning): The experimental procedure of presenting an animal with a reinforcing stimulus immediately following the occurrence of a certain response. Usually, these are responses occurring freely; when the correct response occurs, the animal is immediately reinforced, resulting in an increased rate of responding; e.g., a pigeon may peck at many notes on a piano but when he pecks at the first note of "The Star Spangled Banner," he is immediately reinforced.

OPERANT LEVEL: Base-line or pretesting or prereinforcing level of activity.

OPTICAL-SPREAD FUNCTION: The total effect of the optical processes (diffraction, aberration, light scatter, *accommodation* errors, optical characteristics, and eye movements) by which rays from an external point of light are spread in a bell-shaped distribution curve to form the image on the retina.

OPTIC CHIASMA: The X-shaped junction of the *optic nerves*, a short stout bridge through which, in mammals, there is a partial exchange of fibers between the two nerves. In man and other primates, fibers that originate in the somewhat larger nasal half of the retina pass from one nerve through the chiasma and join fibers in the opposite *optic tract*, whereas the fibers arising from the temporal side of the retina proceed in the lateral portions of the chiasma, without *decussation*, from the optic nerve to the corresponding optic tract. (See Figure 1 in the Introduction.)

OPTIC DISK: The "blind spot" where the layers of the retina are replaced by the *optic nerve* fibers leaving the eyeball.

OPTIC NERVE: Nerve made up of fibers that originate in the *ganglion cells* of the retina and terminate in several paired nuclei (*lateral geniculate nucleus*, pregeniculate gray nucleus) or subcortical stations (*superior colliculus, pulvinar* of the thalamus) in the brain.

OPTIC RADIATION: See *visual radiation*.

OPTIC TECTUM: Dorsal (roof-like) part of the midbrain containing the *superior colliculi*, which form part of the intermediary visual system.

OPTIC TRACTS: The tracts by which the *optic nerves* continue from the *optic chiasma* to the brain. The tract on each side carries fibers conducting impulses set up by contralateral *visual fields* that fall on the ipsilateral retinas, the fibers from the opposite eye having crossed over at the chiasma. (See Figure 1 in the Introduction.)

OPTOKINETIC NYSTAGMUS: Involuntary alternating rapid and slow movements of the eyeball induced by attempting to fixate on a point revolving around an axis.

OPTOKINETIC TEST: A test in which the entire environment appears to move relative to the observer, so that the eyes are compelled to follow the moving object. See also *optokinetic nystagmus*.

OPTOMOTOR NUCLEI: The nuclei of the third, fourth, and sixth cranial nerves, which innervate the *extraocular muscles* and are therefore concerned with the movements of the eyeball.

ORIENTING REFLEXES: A variety of visceral, somatic, neural, and motor responses to initial presentation of a stimulus.

ORTHOPTIC TRAINING: A technique of eye exercises designed to correct the visual axes of eyes not properly coordinated for binocular vision.

OTOLITH ORGANS: Mechanoneural *receptors* in the inner ear that respond to gravitational changes, displacing calcium carbonate crystals (otoliths) attached to the ends of hair cells.

OUTER GRANULAR LAYER: The second layer from the surface of the primate *striate area*, characterized by relatively small cells.

PACING: Gearing of instruction to balance ease and difficulty for a subject learning a task.

PARAFOVEAL RETINA: The area of the retina surrounding the *fovea centralis*.

PARALINGUISTICS: The study of patterned tone of voice.

PATTERNED STIMULATION: Visual stimulation consisting of visual contrast, as opposed to diffuse light.

PERCEPT: A perceived object.

PERCEPTUAL PRETRAINING: Training in the awareness of objects, relationships, or qualities, especially visual, as a preliminary to reading.

PHONEME: The smallest unit of speech that distinguishes one utterance from another.

PHONOLOGY: The science of speech sounds.

PHOTON: A quantum of radiant energy.

PHOTOPUPILLARY MOTION: Reaction of the pupil to light, resulting in changes in its diameter.

PHOTOTONIC BALANCE: Balance in the tonic condition of the intrinsic eye musculature (*ciliary muscle* and *pupillary muscle*) resulting from exposure to particular conditions of lighting.

PLASTICITY: Ability of one part of the brain as a result of experience and training to modify its function to some extent so that eventually it appears to take over the function of a damaged part. Anatomic plasticity refers to the ability of neurons and nerve-tissue aggregations to change by extensions of *dendrites* and interconnections.

PLEOPTICS: A technique of eye exercises designed to develop fuller vision of an *amblyopic* eye and ensure proper binocular coordination.

POLYGRAPH: An instrument for recording tracings of several different events simultaneously.

PONS: A discernible enlargement of the lower brain lying immediately below the midbrain.

POSTSYNAPTIC STRUCTURES: Neurons and their *dendrites* with which efferent terminals of presynaptic neurons make functional contact.

PRAXIS: Motor performance or action, especially that controlled by the cerebral cortex.

PRETECTAL SYSTEM: The system of nerve fibers from the *optic tract* and *superior colliculus* that reach the pretectal nuclei of the midbrain; thought to provide feedback pathways for regulation of eye movements and *accommodation*.

PRIMARY HEALTH SERVICES: First point of contact for medical care or health supervision. In the case of children, these services are provided by pediatricians, general practitioners, public-health or school nurses, or hospital emergency staffs.

PRIMARY STRIATE CORTEX: See *striate area*.

PRIMARY VISUAL SYSTEM: The nerve system that conducts impulses from the retina to the cortical, thalamic, and collicular regions of the brain.

PROPRIOCEPTIVE END ORGANS: Sensory nerve *receptors* that give information concerning movements and position of the body; found chiefly in muscles, tendons, and the *vestibular apparatus*.

PULVINAR SYSTEM: One of several nuclei in the thalamus, a main relay area in the midbrain for sensory input, and having projections to an important association area of the cerebral cortex.

PUPILLARY MUSCLE: The smooth (involuntary) muscle of the iris that controls the size of the pupil.

PYRAMIDAL CELL LAYER: The third layer from the surface of the primate *striate area*, characterized by relatively large pyramid-like cells.

PYRAMIDAL DISCHARGES: Impulses discharged down the pyramidal nerve tracts from the motor cortex and concerned with voluntary motor activities.

REACQUISITION: Regaining, by reinforcement, a conditioned response that has been extinguished.

READING DISABILITY (syn. reading deficiency, dyslexia): A condition in a person whose reading age is below 90% of expectation with respect to his intelligence and educational opportunity. See also *specific reading disability*.

READING RETARDATION: A behavioral term to describe a person who does not score at or above the norm for his age on valid tests of reading.

RECEPTIVE FIELD: A small region of the retina where a spot of light will cause impulses in a single *optic-nerve* or *optic-tract* fiber, in a single *ganglion cell*, or in a single nerve cell in the *visual cortex*. Other single units in these structures may also be activated by the same receptive field or by a different or overlapping retinal receptive field.

RECEPTOR: A sensory nerve terminal that responds to stimuli; in the eye, refers to the *rods* and *cones* in the retina.

RECTI: See *extraocular muscles*.

REDUNDANCY: Superfluous linguistic elements or repetition of cues or information that do not need to be recognized in order to perceive the *Gestalt*.

REFLECTANCE: The reflected fraction of the total luminous flux incident on a surface.

RETICULAR FORMATION (syn. reticular system): A diffuse network or reticulum of nerve cells and fibers, forming the core of the brain stem, that when stimulated produce "arousal." This system is concerned with such general effects as alerting the entire organism, maintaining attention, controlling muscular activity, and regulating receptivity of peripheral sensory end organs.

RETINAL CONTRAST: Physiologic processes in the retina that produce noticeable subjective differences between objects of different luminosities placed side by side.

RETINAL RIVALRY: A condition in which one area of the retina is dominant and the corresponding retinal area of the other eye, recessive; spontaneous reversal of the condition is implied.

ROD: A photoreceptor neuron characterized in part by an elongated shape that synapses with some *bipolar cells* and is used chiefly in night vision; anatomically absent from the *fovea centralis*; in man, a fairly uniform cylinder 24 μ long and 3 μ wide.

RUBIN'S GOBLET FIGURE: An ambiguous figure that can be seen either as a goblet or as two facial profiles.

SACCADE: A jerk-like movement of the eyes horizontally in either direction as the *fixation* point changes, interspersed with pauses. A skilled reader will demonstrate smooth movements in one direction with few pauses and a regressive movement at the end of a line.

SCHEMA (pl. schemata): A plan, outline, or arrangement.

SCOTOMA: A blind spot; an area of depressed vision within the *visual field*, due to retinal damage or to specific lesion within the visual pathways or *visual cortex* and surrounded by an area of less depressed or normal vision.

SEGMENT (syn. chunk): To break up continuous spoken sound into segments on the basis of knowledge of the speaker's language.

SEMICIRCULAR CANALS: Within the *vestibular apparatus*, three canals each in a different plane at right angles to each other, and sensitive to movement of the endolymph contained within them due to change in position, acceleration, or convection.

SEPARATION ANXIETY: The fear and apprehension noted in infants when removed from their mothers (or surrogates) or when approached by strangers.

SHADOWING: Repetition of an auditory stimulus slightly after the original presentation.

SHORT-TERM MEMORY: Retention of information for about a minute or less.

SNELLEN FRACTION: A measure of visual acuity based on Snellen's test type; the numerator is the subject's distance from the test type, and the denominator, the distance at which the letters could be read by a normal eye. Thus 20/20 means normal vision in the eye being tested.

SOMADENDRITIC FACTOR: In the neuronal tissue (soma), an undefined factor that determines the distribution of *dendritic spines* along the *dendrites*.

SOMATESTHESIA (syn. somesthesia): The consciousness of having a body.

SOUND SPECTOGRAPH: An instrument that portrays in graphic form the time variations of the frequency spectrum of the speech (sound) wave.

SPECIFIC READING DISABILITY (syn. developmental reading disability, congenital reading disability, strephosymbolia, specific dyslexia, congenital dyslexia): A condition in which the reading age is below 90% of expectation with respect to measured intelligence and educational opportunity, but with no evidence of structural defect of the central nervous system and with peripheral sensory apparatus sufficiently intact for the recognition of sensory stimuli involved in reading. There is evidence that this is due to an inherited lag in the maturation of neurophysiologic functions (as yet unidentified) involved in reading.

SPECIFIC VISUAL AFFERENTS: Afferent fibers conducting nerve impulses from the visual photoreceptors (*rods* and *cones*) to the brain.

S-R: Stimulus–response; usually refers to theories of the acquisition of information and learning.

STABILIMETER: Apparatus for recording the amplitude and frequency of the motions of a subject.

STELLATE CELLS: Star-shaped cells found chiefly in *layer IV* of the *striate area* and characterized by the horizontal arrangement of their processes.

STEREOGNOSIS: Ability to perceive and recognize shape or form qualities of an object by handling it; tactile recognition of form.

STEREOPSIS: The sensation of relative visual depth that results from the neural integration of dissimilarities in the images seen by the two eyes.

STRABISMUS: Deviation of the eye that a subject cannot overcome; the visual axes of the two eyes assume a position relative to each other different from that required by the visual stimulus conditions.

STRATIFIED RANDOM SAMPLING: Random drawing of individuals from each of several strata of the population, in contrast to sampling by chance alone.

STREPHOSYMBOLIA: Perception of visual stimuli, especially words, in reverse order but without the reversal of individual letters characteristic of mirror perception.

STRIATE AREA (syn. area 17, area striata, visual I, primary striate cortex): The primary visual receiving area or highest brain center of vision. A portion of the cerebral cortex in the inner, medial space of the *occipital lobe* of each cerebral hemisphere, where the fibers of the *visual radiation* terminate.

SUBJECTIVE CONTRAST: The subjective experience produced by physiologic retinal and brain contrast mechanisms, distinguished from contrast in the visual stimulus itself.

SUPERIOR COLLICULAR SYSTEM: The nerve system in the forward or rostral pair of the two pairs of rounded eminences (colliculi) in the roof of the midbrain concerned primarily with regulation of ocular reflexes and adjustment of head and body position relative to visual orientation.

SUPPRESSION: Overriding of one response by another, as distinct from inhibition of a response. In visual suppression, the specific mechanisms are varied and unknown, and the visual input in an eye may be entirely or partially blocked.

SUPRAGRANULAR LAYERS: Layers I-III lying above or toward the surface from *layer IV* of the primate *striate area,* which is characterized by granular cells.

SUPRANUCLEAR LESIONS: Lesions of higher (i.e., more centrally located) structures than the *nucleus* associated with the nerve pathway involved.

SURFACE STRUCTURE (syn. surface form): Elements of a sentence that determine its phonetic interpretation, including the actual formatives, phonetic signals, and combination of perceived utterances.

SURROUND FIELD: An area surrounding a center or *receptive field* where both on and off responses are obtained to light stimulation.

SYNAPSE: Locus at which a nerve impulse passes from the *axon* of one neuron to the *dendrites* of another; forms boundary between the two nerve fibers or possibly only a surface of contact.

SYNTACTIC REDUNDANCY: Repetition of elements of syntax, the rules by which sentences are constructed.

TACHISTOSCOPE: An instrument for the very brief presentation of visual stimuli, such as dot patterns, words, numbers, or pictures of objects; it is capable of presenting stimuli to either eye at intervals of less than 0.001 sec.

TEMPORAL LOBE: The lower lateral lobe of the cerebral hemisphere, merging behind with the *occipital lobe* and subserving the hearing and equilibration senses.

TRANSNEURONAL DEGENERATION: Degeneration or atrophy of a neural pathway or structure when the neurons with which they make synaptic connection are cut, or in some cases when the end organs and their afferent nerves are not used.

TRIGGER FEATURE: A particular set of stimulus conditions capable of eliciting activity in a single unit in the retina or higher visual center.

TROLAND: A unit of intensity of light at the retina equal to the illumination received per square millimeter of a pupillary area from a surface having a brightness of 1 candle per square meter.

VESTIBULAR APPARATUS: The system of intercommunicating *semicircular canals* and the *otolith organ* within the inner ear (filled with endolymph) that forms a sense organ stimulated by gravity and rotational movements.

VESTIBULAR MOVEMENTS: Reflex movements of the eyes initiated by impulses from the *vestibular apparatus* in the inner ear due to rotation of the head.

VISUAL CLIFF: A sharp vertical discontinuity in visual space, such as the edge of a desk, either real or contrived by optical illusion.

VISUAL CORTEX: See *striate area.*

VISUAL DISCRIMINATION: The differentiation between visual patterns.

VISUAL FIELD: What a fixating eye can see. Visual fields for the two eyes are normally somewhat ovoid and overlap.

VISUAL-MOTOR: Refers to the coordination of vision with movements of the body or of parts of the body.

VISUAL NOISE: A visual stimulus having no recognizable organization, such as scattered parts of letters or numerals.

VISUAL PLACING: A visually directed response consisting of placing of one or more of the extremities upon an object.

VISUAL PROJECTIONS: Nerve fibers carrying visual impulses that originate in the retina and are directed or relayed to other parts of the brain.

VISUAL PROJECTION AREAS: Cortical areas, such as the *striate area*, to which fibers from the *lateral geniculate nucleus* project and transmit impulses.

VISUAL RADIATION (syn. optic radiation): The paired nerve tracts or fiber system carrying visual impulses from the *lateral geniculate nucleus* to the highest brain center of vision, the *striate area.*

VISUAL RESOLVING POWER: A measure of visual acuity or the ability of the eye to discriminate or resolve small visual stimulus differences.

VISUAL II AREA: See *area 18.*

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